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THE GASTROINTESTINAL RESPONSE OF CHILDREN TO TEST MEALS OF BARIUM AND PASTEURIZED, EVAPORATED, AND BASE-EXCHANGED MILKS

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IN ANY study of nutrition and growth of children it is important to consider the movements and motility of the alimentary canal, the secretions which reach it, the chemical changes which proceed within it, and the absorption and utilization that take place; as well as the general fitness and response of the body as a whole. Indeed, Mendel[1, 2] cogently pointed out three and two decades ago, "The relation which exists between the functions of the digestive tract and that ensemble of biologic processes termed nutrition is both manifold and far-reaching," and that in the considerations of the digestive organs one "cannot fail to find some interest in the role of foods, for the transformation and transport of which these organs exist, or in the phenomena of nutrition, for which the function of digestion is an indispensable preparation." It is important, he states further, "to observe the conditions of interdependence of cooperation between different physiologic tissues;" and to recognize that "the study of alimentation is something more than the mere consideration of the solvent action which the digestive secretions can exert, but is the consideration of the cooperative working of many functions—an orderly march of events."

The digestion and absorption of food depends upon the type and condition of the bolus of food in the stomach, and subsequently in the alimentary tract, and the length of time it is exposed to the various types of digestive and absorptive processes. Brennemann, in his classic work on the coagulation of cow's milk in the stomach[3] and the buffer action in infant feeding,[4] has demonstrated that cow's milk in the human stomach is a peculiarly solid food; that the coagula from it become larger for about two hours after formation, by agglutination, and thereafter continuously harder, by contraction, and that digestion occurs at their periphery only. Modern infant feeding and treatment for certain types

From the Research Laboratory of the Children's Fund of Michigan in cooperation with Harper Hospital and the Methodist Children's Village.

of underpar individuals has indicated a need for the reduction in size and consistency of the usual curd of natural cow's milk. In the preparation of the proprietary or therapeutic milks an attempt is made to accomplish this and thereby obtain a modified milk whose curd more nearly approaches that of breast milk, by correcting either the curd or the pH to increase the ease of digestion with a resultant increased efficiency in utilization. Emphasis has been placed on preheating alone, thereby improving tryptic digestion[5] and denaturization of protein; preheating and homogenization, as in the preparation of evaporated milk; lowering the pH value and thereby favoring peptic activity[6] and gastric motility, as well as stimulating pancreatic secretion by the addition of acid to the milk formula. The subject of this investigation is a study of the comparative gastrointestinal motility rates of children when given pasteurized, evaporated, and a processed, "base-exchanged" milk[7] specially treated to bring about a base exchange by passing it through sodium-aluminum silicate (zeolite).

The term "soft curd milk" has been given to milks which, when treated with rennin, form a softer, more flocculent curd resembling breast milk, in contrast to the tough curd of regular cow's milk. Recent metabolic balance studies have indicated some of the advantages of certain soft curd milks such as evaporated milk in the feeding of both infants[8] and children[9, 10] and, in infant feeding, milk that has been filtered through a bed of zeolite to alter its mineral distribution.[11] Numerous clinical studies of the human response to different kinds of soft curd milks have produced varying results. Since, in our experience, evaporated milk gave a larger storage of nitrogen, sulfur, and potassium and stimulated soft tissue growth in children,[9] the decision was made to test the response of the same subjects, whose gastrointestinal patterns were known,* to various types of milk to determine whether milks of different curd tensions alter gastrointestinal motility rates and are, in this way, a contributing factor to more efficient utilization.

Hill[12] has demonstrated that the curd tension of milks (the pull in grams required to draw a special star-shaped knife through a curd formed by adding a solution of rennin to milk) may vary from 15 to 200 gm.; on the average, 60 gm. for ordinary cow's milk. Pasteurization at 143° F. for one-half hour has little effect upon the resistance of the curd formed by the coagulation of milk with pepsin. Any milk with a curd tension below 30 gm. is considered a soft curd milk. Both evaporated and base-exchanged milks fall into this classification; the curd tension of the first being reduced by heat and homogenization and that of the latter by percolating the milk through zeolite, thus reducing the calcium, phosphorus, and potassium contents and increasing the sodium value.

*The gastrointestinal patterns of growing children will be treated as a section of a monograph upon the chemistry of growth. The subjects of this study were among those upon whom metabolic studies were carried over eight consecutive months to accumulate part of the monograph material.

Espe and Dye[13] boiled a raw milk of 117 gm. curd tension and reduced its curd tension to 5 gm. They also demonstrated that doubling the curd tension of milk increased the length of the digestive period from 30 to 65 per cent. All investigations have seemed to indicate a closer relation of the protein in the milk to the curd tension than to the fat dispersion. The evaporated milk which is not only heated but is also homogenized produces a higher nitrogen utilization in infants[8] and children,[9, 10] as shown by metabolic balance data, and it is comparable to breast milk in the digestive tract of premature infants. Poole and Cooley[14] gave each of five premature infants a test meal of breast milk and barium, and, on another occasion, a test meal of nonacidified evaporated milk-barium formula and saw little difference among roentgenograms showing the gastric emptying times of the two milk formulas. A considerable part of each meal left the stomach within the first half hour and there was little remaining in the stomach after three hours.

EXPERIMENTAL

The subjects of this research, one girl and six boys, were normal healthy children aged 7 to 11 years, who had participated in biologic studies for several years; they were experienced subjects accustomed to laboratory procedures, medical and physical examinations, and metabolic routine, in all of which they were interested and therefore cooperative. The same children had been the subjects of several previous roentgenologic studies. Indeed, the emotional factors were so minimal that there was wholesome competition among the members of the group, each child endeavoring to excel in facilitating the accuracy and speed of the study. Minute scrutiny of their medical histories had failed to reveal any known detrimental factors due to heredity, idiosynerasy, or previous illnesses.

Every effort was made to keep all experimental conditions as uniform as possible. The children lived together, with a housemother, in a home in the country which provided a healthful and happy environment.[9] The dietaries consumed were made up of foods which the children enjoyed and were known to have adequate nutritive value. The food essentials and bulk remained approximately constant from day to day. To eliminate questions of adjustment to a particular type of milk, the milk to be studied was incorporated into the diet one week preceding the roentgenographic study. Regular habits of activity, sleep, eating, and elimination had been established prior to, and were maintained throughout, these gastrointestinal studies. A daily record was kept of the number of bowel movements of each child during the entire 22 days of this study to determine whether there was any change in the rhythm of the subjects' elimination in response to the different types of milk.

The observations were made during the month of October when the stomach's response is said to be most active. Todd[15] states that the stomach during late winter and early spring is not so active. Weather conditions were favorable for optimal health and out of door activity in the sunshine. The United States Weather Bureau records for the 22 days show that the mean temperature per day was 59° F., range 42-71°; total precipitation, 0.23 inch; and average daily sunshine 9.2 hours. Fourteen days were clear; five, partly cloudy; and three, cloudy.

The milks used as the media for barium sulfate in the test meals originated from the same source.* The whole milk was collected from one group of cows and consequently all the milks originally had the same curd tension. One week's supply was pasteurized, another was evaporated, and a third was slightly acidified, then percolated through a zeolite bed; each particular type of milk was prepared under the usual commercial conditions. The chemical composition of the various milks used in the study is given in Table I.

TABLE I

COMPOSITION OF MILKS

	PASTEURIZED MILK	EVAPORATED MILK (DILUTED 1:1)	BASE-EXCHANGED MILK
	GM./100 GM.	GM./100 GM.	GM./100 GM.
Butterfat	3.55	4.09	3.50
Total solids	12.10	13.75	12.00
Protein	3.21	3.45	3.21
Ash	0.74	0.88	0.70
Calcium	0.121	0.135	0.100
Magnesium	0.013	0.015	0.014
Sodium	0.052	0.067	0.096
Potassium	0.171	0.167	0.117
Phosphorus	0.097	0.108	0.083
Chlorine	0.113	0.124	0.106
Sulfur	0.050	0.051	0.046

It may be noted that the evaporated milk when diluted 1:1 with water was slightly more concentrated in all constituents than the original milk, as is usually true, due to the removal of 60 per cent of the water content by the evaporation process. The base-exchanged milk gave up approximately 17 per cent of its calcium, 14 per cent of its phosphorus, 6 per cent of its chlorine, and 32 per cent of its potassium in exchange for an increase of 85 per cent in sodium and 7 per cent in magnesium content.

The empty stomach has the ability of accommodating itself to the size of the meal received in it, consequently the stomachs of all normal children within a narrow age range would appear approximately the same size. In this study each test meal consisted of 2 ounces of barium sul-

*Mr. C. G. Reichle, plant manager of the Detroit Branch of the Borden Farm Products Company of Michigan, skillfully controlled the handling and preparation of all the milks used in this study.

TABLE II

EMPTYING TIMES OF THE STOMACHS OF SEVEN CHILDREN IN RESPONSE TO TEST MEALS OF BARIUM AND PASTEURIZED, EVAPORATED, AND BASE-EXCHANGED MILKS

SUBJECT	AGE (MO.)	SEX	RECUMBENT LENGTH (CM.)	WEIGHT (KG.)	PHYSICAL* TYPE	BARIUM AND PASTEURIZED MILK		BARIUM AND EVAPORATED MILK		BARIUM AND BASE-EXCHANGED MILK	
						NUMBER OF ROENTGENOGRAMS	STOMACH EMPTYING TIME (MIN.)	NUMBER OF ROENTGENOGRAMS	STOMACH EMPTYING TIME (MIN.)	NUMBER OF ROENTGENOGRAMS	STOMACH EMPTYING TIME (MIN.)
D. P.	135	M	152.0	39.92	0.0226	9	195	8	180	8	150
W. P.	131	M	132.0	35.27	0.0248	10	240	9	220	8	150
H. H.	120	M	139.0	32.43	0.0229	10	240	9	240	9	195
F. C.	114	M	134.0	30.39	0.0232	9	180	7	210	9	285
R. S.	102	M	132.6	26.42	0.0224	9	240	8	240	8	210
B. M.	99	F	123.2	23.81	0.0234	9	255	9	240	9	210
J. H.	82	M	125.5	25.86	0.0236	9	240	8	165	7	150
		Total				65		58		58	
		Average					*227*		*214*		*193*

* $\sqrt[3]{\frac{\text{weight}}{\text{height}}}$

fate in 4 ounces of milk, thoroughly emulsified with an electric mixer and served at body temperature. Neither food nor liquid was taken between the evening meal and the time of the barium-milk meal about ten o'clock the following morning. These two factors were held constant, since it has been shown that there is a definite relation between temperature,[16] the size of the meal,[17] and gastric emptying time. Previous roentgenographic examinations had eliminated the possibility of abnormalities in the lungs, heart, or thymus of any of the subjects.*

RESULTS AND DISCUSSION

Table II shows the age, size, and physical type of the subjects, the number of roentgenograms exposed during the progress of each test meal, and the emptying time of the stomach in response to each type of milk. In Fig. 1 the progress of the three different types of milk test meals through the tracts of the seven children is shown pictorially. Necessarily, the representations in the figure are approximate and show progress only through the anatomical sections of the gastrointestinal tract which can be clearly distinguished in the roentgenograms; this method of symbolizing is not quantitative in the sense that the volumes of any test meal in any section of the tract are indicated by the area representing them, nor are the sections of the tract equal in length or volume as they are portrayed.

In Table II the average gastric emptying times are shown to be less for the soft curd milks (214 and 193 min.) than for the pasteurized milk (227 min.). The average gastric emptying time of the base-exchanged milk (193 min.) is less than that for the evaporated milk (214 min.); however the range is narrower for the evaporated milk (165-240) than for the base-exchanged milk (150-285). Among the individuals, six of the seven present the same relationship between the different test meals as that shown by the averages. One child, F. C., shows a response opposite to that of the other subjects.

Ten minutes after ingestion of the pasteurized milk meal, the roentgenograms of six of the children showed the milk meal remaining in the stomach (Fig. 1). Upon the first exposures of the evaporated milk series four subjects' stomachs had started to empty; on those of the base-exchanged milk series five had released some of the meal from the stomach; three of the latter series and one of the former indicated some of the test meal was in the jejunum.

At the end of one hour after ingestion of the meals the more rapid motility of the soft curd milk meals was very definite and is easily seen in Fig. 1. After two and three hours the soft curd milks show greater advancement through the intestinal tract, but it is not as pronounced as at the one-hour interval, i.e., the pasteurized milk meal made rela-

*See footnote on p. 2.

tively greater progress during the interval between one and three hours after ingestion of the meal. Twenty-four and forty-eight hours after ingestion of the meals, roentgenograms showed the same characteristics

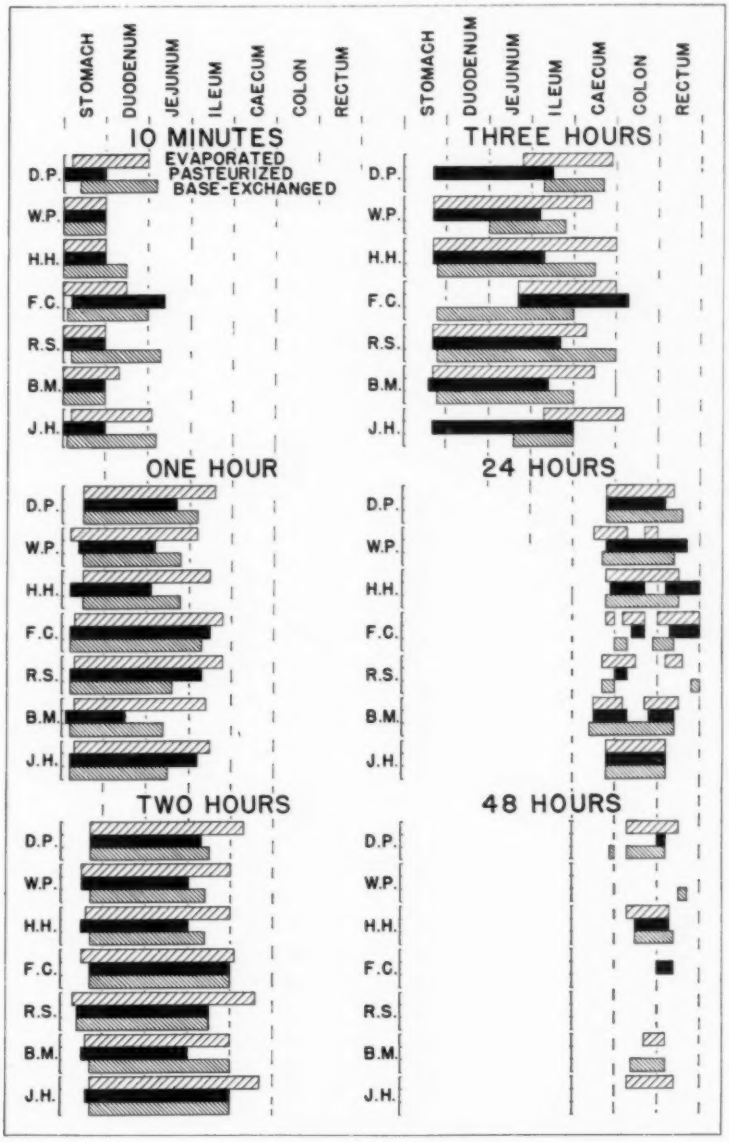

Fig. 1.—Schematic representation of the progress through the gastrointestinal tracts of seven children at comparative time intervals of test meals of barium sulfate and evaporated, pasteurized, and base-exchanged milks.

which were presented in the study of the gastrointestinal patterns of the same children.*

These results indicate that, since the soft curd milks passed so rapidly out of the stomach and into the intestine, there was less opportunity for the first portions of the soft curd milks to be exposed to the acid and enzymes of the gastric juice; on the other hand, the delayed emptying time of the stomachs in the case of the pasteurized milk, allowed the food bolus a longer exposure to peptic digestion. The soft curd milks were subjected for a longer time to digestive juices of the upper small intestine, and there was either a slowing down in the rate of passage of these milks through the duodenum, jejunum, and ileum, or there was a speeding up of the pasteurized milk through this portion of the alimentary canal. It is possible that the curd of pasteurized milk, with its longer exposure to peptic digestion, was thereby partially broken down and tryptic digestion could proceed at a more rapid rate in the upper small intestine. The more complete the digestion the more rapidly absorption can take place. The fact that the pasteurized milk remains in the stomach for a longer period of time initiates numerous inquiries: is it due to a different buffering effect that the various milks have in the stomach; is it due to the physical condition of the fat, the fat contents being approximately the same; or, is it due to the physical condition of the curd and the delayed peptic digestion?

The contrast between the films of the modified milks and those of the pasteurized milk is marked and shows the modified milks to be in fluffier masses and more evenly dispersed throughout the tract, and in the colon and rectum they do not appear as compact and solid. A representative example of the differences in response to the three types of milk meals is presented in Fig. 2, in which roentgenograms at equivalent time intervals from each series of roentgenograms of one subject (J. H.) are reproduced. The faster gastric emptying times with the processed milks may be seen easily; likewise, the softer, more flocculent masses of the soft curd milks appear in contrast to those of the pasteurized milk.

There is, apparently, some difference between the reactions to the soft curd and the evaporated milks. There seems to be a tendency for an orderly diffusion in the progress of the meals from the stomach and through the rest of the tract, but this progression, seemingly, is initiated earlier with the evaporated milk than with the base-exchanged milk but is more rapid with the latter, therefore producing approximately the same relative motility over all the tract. In several of the children it would seem from the twenty-four- and forty-eight-hour films that the modified milks were retained for longer periods in the intestines than the pasteurized milk, but that the pasteurized milk may be retained longer in the rectum. The base-exchanged milk contains less calcium than the pasteurized or evaporated milks, and there would be a reduced tend-

*See footnote on p. 2.

ency to form the insoluble soaps of fatty acid in the large intestine. Pasteurized and evaporated milks carry about the same amount of calcium; therefore the excretion of soaps and fat would be about the same and might have a tendency to remain longer in the lower colon.

During the four days preceding each test meal which was studied roentgenologically, during which time the same type of milk was included in the daily diet, the average numbers of defecations were as follows: the four days before pasteurized milk test meal, 1.4 per child per day; before the evaporated milk test meal, 1.5 defecations per child per day; and before the base-exchanged milk test meal, 1.4 defecations per child per day. The average number of stools per child per day, over the twenty-two-day period within which the three test meals were given, was also 1.4. While the test meals were studied at intervals of one week, the first three days, during which the exposures were made, are not included in the averages of the defecations, due to the possible influence of the changes in bulk ingested, and the break in routine produced by the roentgenologic study. In addition, during the three days of each study, it is known that a constipating effect of the barium indicator would be portrayed in the frequency of eliminations.[18] This factor is also indicated by the average of 1.4 bowel movements per child per day during the 22 days within which three barium test meals were given, in contrast to the average of 1.8 stools per child per day during the previous metabolic study of the same children over eight consecutive months, during which no barium was given and the same standardized conditions of diet, habit, routine and environment were maintained.*

SUMMARY

To determine the relation of gastrointestinal motility to milks of different curd tension, seven healthy children, aged 7 to 11 years, each of whom had been observed previously in an acid-base mineral balance study covering 225 successive days, and whose gastrointestinal motility patterns were known, served as subjects of serial roentgenographic studies upon the orderly passage through the alimentary tract of test meals composed of 2 ounces of barium sulfate and 4 ounces of pasteurized milk, evaporated milk diluted 1:1 with water, and base-exchanged milk. Roentgen examinations were made at frequent intervals until the meal had passed out of the stomach and at twenty-four-, forty-eight-, and seventy-two-hour intervals after its ingestion, a total of 181 exposures.

The average gastric emptying times for the seven children were 227, 214, 193 minutes, respectively, for pasteurized, evaporated (diluted 1:1), and base-exchanged milks. The roentgenograms taken ten minutes after ingestion indicated that the soft curd milks (evaporated and base-exchanged) began emptying from the stomach in much less time than the pasteurized milks: in nine instances the soft curd milk had reached

*See footnote on p. 2.

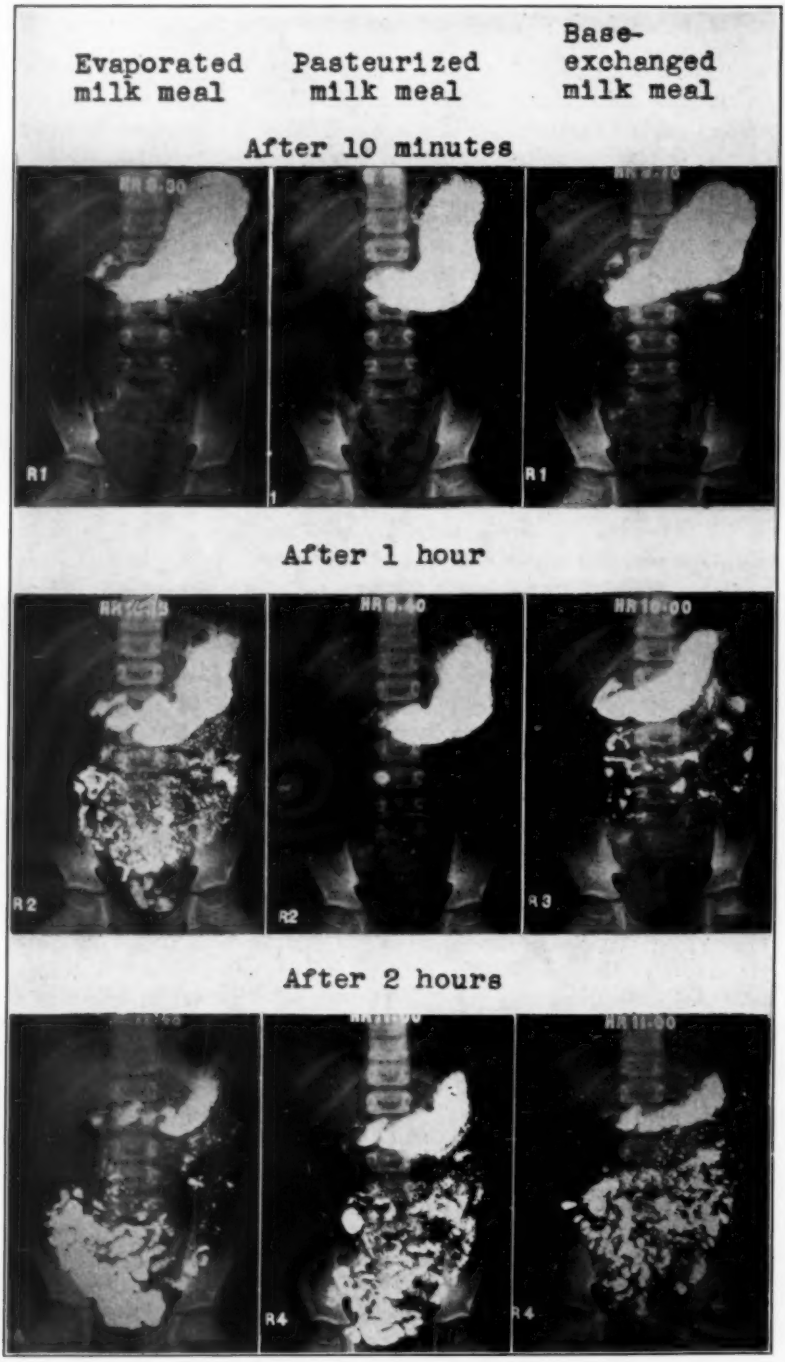

Fig. 2.—Roentgenograms of progress of test meals of barium sulfate and evaporated, pasteurized, and base-exchanged milks, through the gastrointestinal tract of one child (J.H.).

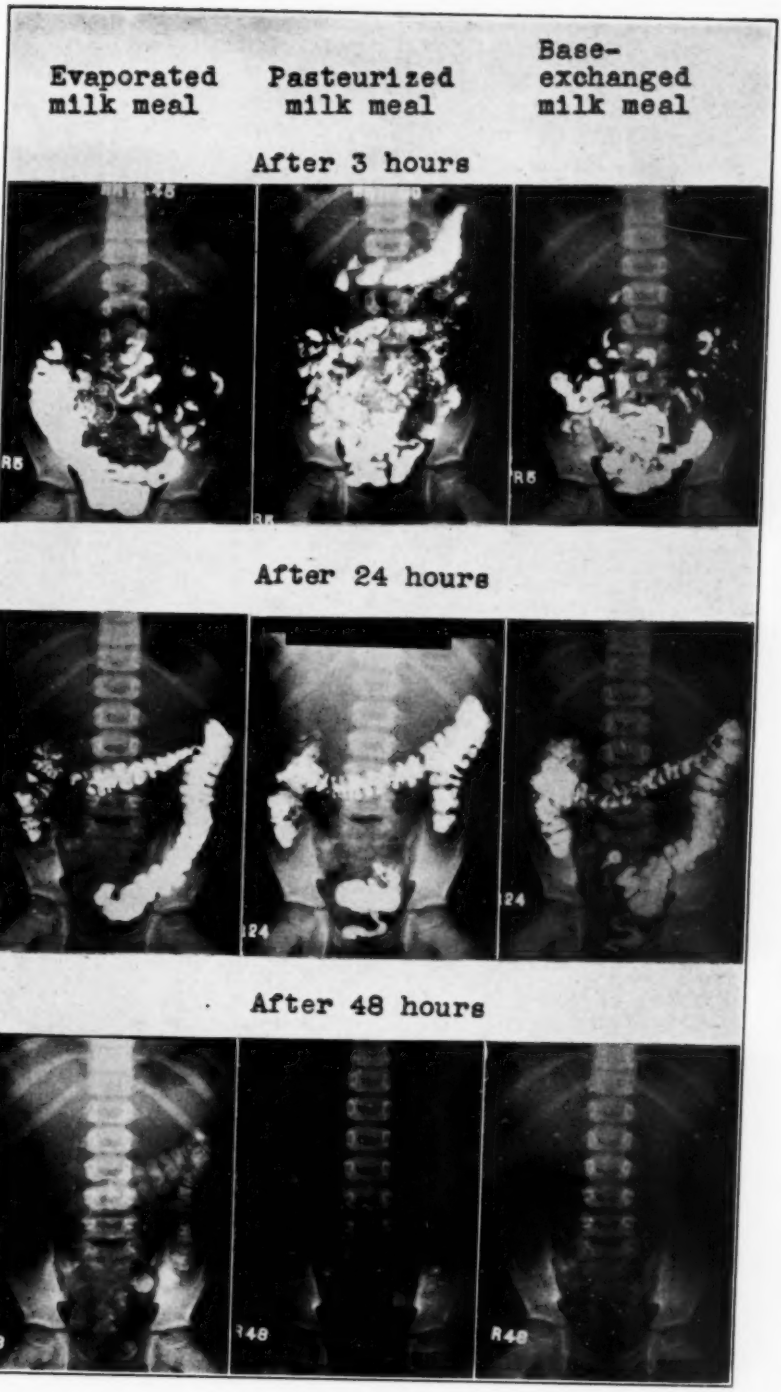
Evaporated milk meal
Pasteurized milk meal
Base-exchanged milk meal
After 3 hours
After 24 hours
After 48 hours

Fig. 2.—Cont'd.

the duodenum and in four of these had entered the jejunum after ten minutes, while in only one exposure at the 10-minute interval had the pasteurized milk meal left the stomach.

The fluffier, more evenly dispersed masses formed by the processed milks, presenting greater surface areas to the digestive juices in the small intestine, and the more orderly progression of these soft curd milks throughout the alimentary canal, are in accord with results of metabolic and clinical observations upon the nutritive advantages of different milks.

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CONGENITAL SYPHILIS

I. Routine Treatment With Acetarsone of Infants Whose Mothers Were Inadequately Treated During Pregnancy

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SUCCESS in the treatment of congenital syphilis is closely related to the time at which it is instituted. Antisyphilitic therapy should be administered to every infected pregnant woman and, if indications exist, continued throughout the first year of the life of the infant. A review of the experience in the five children's venereal clinics under our control emphasized (a) the inadequacy of prenatal treatment, (b) the failure of parents to cooperate in the treatment of their children, and (c) delay in the initiation of treatment in many syphilitic children.

A. INADEQUATE PRENATAL TREATMENT

Syphilis was diagnosed in approximately 5 per cent of the white women and in 16 per cent of the negroes among 3,000 consecutive obstetric patients in the Cincinnati General Hospital. In checking the records of 187 syphilitic mothers observed over a period of one and one-half years it was found that only 25 had received antisyphilitic treatment during pregnancy and that only six of this number had received adequate treatment, that is, for more than four months (Table I).

Table I

Antisyphilitic Treatment During Pregnancy

	Number of Women	Per Cent
No treatment	162	87
Irregular, sporadic	13	7
Treated less than four months	6	3
Treated more than four months	6	3

B. FAILURE OF COOPERATION OF PARENTS OF POTENTIALLY SYPHILITIC INFANTS

Every syphilitic mother was requested to return to the nearest clinic with her newborn infant at least within six weeks after discharge from the obstetric wards, and nurses visited her at her home to urge such attendance. In spite of this, a large number of mothers did not return (Table II).

From the Department of Pediatrics, University of Cincinnati, and the Children's Hospital Research Foundation.

TABLE II

NUMBER OF SYPHILITIC MOTHERS WHO RETURNED TO CLINIC AND THEIR COOPERATION IN THE TREATMENT OF THEIR INFANTS

Number who returned to clinics	109		60.6%
Good cooperation at clinic		58	
Fair cooperation at clinic		10	
Poor cooperation at clinic		41	
Number who did not return	71		39.4%
Because of indifference		25	
Because of refusal of treatment		12	
Degree of cooperation not determined because of death of baby (pneumonia, lues, accident)		5	
Treated by private physician		4	
Changed residence without notification		25	
Total	180		

C. DELAY IN BEGINNING TREATMENT

Of 300 cases of congenital syphilis treated in our clinics during the past ten years, 64.3 per cent had no symptoms of the disease of sufficient severity to cause any alarm on the part of the parents. The diagnosis had been made in these cases by serologic tests performed because of the history of the disease in the family, or as a routine measure when the children were admitted to institutions or hospitals. The age at which the disease was detected and the factors leading to the diagnosis of syphilis have been listed in Table III.

TABLE III

FACTORS LEADING TO THE DIAGNOSIS OF SYPHILIS IN 300 CHILDREN WITH POSITIVE SEROLOGIC TESTS AND THE AGE AT WHICH TREATMENT WAS STARTED

AGE (YR.)	NUMBER OF PATIENTS	ACTIVE SYMPTOMS OF SYPHILIS		HISTORY OF SYPHILIS IN FAMILY		ROUTINE SEROLOGIC TESTS	
		NO.	%	NO.	%	NO.	%
Under 1	69	29	42.0	27	39.1	13	18.8
1	22	7	31.8	9	40.9	6	27.3
2	21	5	23.8	7	33.3	9	42.9
3	16	4	25.0	5	31.3	7	43.8
4	17	4	23.5	6	35.3	7	41.2
5	26	9	34.6	10	38.5	7	26.9
6	13	2	15.4	2	15.4	9	69.2
7	24	4	16.7	10	41.7	10	41.7
8	11	5	45.5	2	18.2	4	36.4
9	17	7	41.2	4	23.5	6	35.5
10	12	6	50.0	2	16.7	4	33.3
11 to 14	52	25	48.1	10	19.2	17	32.7
Total—all ages	300	107	35.7	94	31.3	99	33.0

This table shows that approximately one-third of the syphilitic children seen under 10 years of age and one-half of those seen first between 10 and 15 years of age developed clinical symptoms of the disease.

The high percentage of patients with latent syphilis may be due to failure to recognize minor symptoms, the parents not considering them

as indicating more than a passing illness. Sporadic but insufficient antisyphilitic treatment received by a number of mothers may account for the mildness of symptoms in some cases.

The detection of syphilis in the symptom-free children is dependent upon routine and repeated serologic examinations—a program of considerable magnitude. It is important that the diagnosis be made in the early weeks of life so that treatment may be instituted at a time when it is most effective. It is unfortunate that during the neonatal period an infant may not exhibit clinical manifestations of syphilis and that the serologic test at this time may be inconclusive, the latter difficulty arising, of course, because many syphilitic infants have negative serologic tests and because some healthy infants have temporary positive reactions due to antibodies transmitted to them from their mothers. Aids in making the correct diagnosis include roentgenograms of the long bones, quantitative serologic tests, and the examination of the cord for spirochetes.

Treatment.—Until seven years ago (1932) we employed sulpharsphenamine and neoarsphenamine in combination with injections of mercury or bismuth in the treatment of children with congenital syphilis. Recently acetarsone by mouth has been used in some of our clinics in combination with mercurial inunctions. The active lesions of congenital syphilis have seemed to respond well to this therapy, and the cooperation and willingness of the parents to bring their infants for treatment have improved.

The results obtained have been published elsewhere.[1] Ten (67 per cent) of a group of 15 infants who were less than two years of age when treatment was instituted responded satisfactorily, as shown by reversal of their serologic tests and freedom from symptoms by the end of one year. With sulpharsphenamine and neoarsphenamine 15 (79 per cent) of a group of 19 infants showed similar results. In older children, 2 to 15 years of age, the results were less satisfactory since negative serologic tests and freedom from symptoms occurred in only 13 (34 per cent) of a series of 38 children treated with acetarsone, and in 13 (21 per cent) of 62 children treated with other arsenicals.

We were so impressed with the importance of giving treatment early in life that we advised routine treatment to a group of newborn infants whose mothers were syphilitic and who had not received adequate antisyphilitic treatment during pregnancy. The majority of these mothers had had little or no treatment, and none had received more than four months of regular treatment. The infants were treated with daily mercurial rubs during the first week of life. Appointments for the venereal disease clinic were given to the mothers when they left the hospital, and a nurse visited the patients at their homes to

see that the inunctions were being continued and that the infant was brought to the clinic at 4 to 6 weeks of age. At that time the rubs were discontinued, acetarsone treatment was instituted, and the infants were observed at weekly intervals.

It was planned to administer treatment for one year regardless of the serologic reactions. At the end of this time, another continuous year's treatment was to be given if the serologic test was still positive. The patient was then to be observed at regular intervals and about two courses of treatment were to be given yearly unless the symptoms or serologic tests indicated otherwise.

A group of 36 newborn infants of syphilitic mothers were treated for at least one year according to this plan; about one-third were white infants and the remainder were negroes. As already stated, mercurial inunctions of 5 to 10 grains were started while the infants were in the hospital and the mothers were instructed to continue the treatment at home during the first six weeks. All but 5 of these patients returned to the clinic and therefore started on acetarsone treatment before they had reached the age of 2 months. Treatment was delayed until the age of 3 or 4 months in 4 patients and until 8 months in 1 patient.

In 27 of these 36 infants serologic reactions were obtained on the cord blood; 22 showed positive tests. Symptoms suggestive of syphilis, which occurred in 11 infants, consisted of snuffles or skin lesions. These manifestations disappeared in one to five weeks after treatment was started. After treatment for one year, all of the patients were free from symptoms and had negative serologic reactions except the 1 patient whose treatment had been started at 8 months of age; in this instance the reaction was positive to a sensitized antigen only. Ten of the patients have been observed for one to three years since the termination of the treatment, and they have remained free from symptoms of syphilis and still have negative serologic reactions.

TABLE IV

INFANTS BORN OF INADEQUATELY TREATED SYPHILITIC MOTHERS

TOTAL NUMBER	NO INITIAL SYMPTOMS	SYMPTOMS OF SNUFFLES, SKIN RASH	CORD SEROLOGY			RESULTS AFTER 1 YR. OF TREAT.	
			NEG.	POS.	NOT TAKEN	NEG. SEROLOGY, NO SYMPTOMS	DOUBTFULLY POS. SEROL. NO SYMPTOMS
36	25	11*	5	22	9	35 (97.2%)	1 (2.8%)

*In 7 of this group, the serologic tests of the cord blood were positive, in 3 negative, and in 1 no test was made.

Complications of the treatment occurred in 13 infants, consisting of diarrhea in 8 instances, vomiting in 5, stomatitis in 2, skin rash in 1,

and an intermittent albuminuria in 1; all of these promptly ceasing when the treatment was discontinued for a short time and not recurring when it was reinstituted.

The most satisfactory method of preventing congenital syphilis is, of course, the prevention of the disease by adequate treatment of the mother during pregnancy. Second in importance is the routine treatment of all newborn infants whose mothers had not had adequate treatment.

Routine treatment of the newborn infants of syphilitic mothers has been proposed by a number of writers.[2] It can be argued that the treatment of the pregnant syphilitic woman is directed toward treatment of the fetus and that, if there is delay in treatment of the infant after its birth until an absolute diagnosis is made, the chances of syphilitic manifestations will be increased. Furthermore, if the mother is so indifferent that she does not cooperate to secure adequate treatment during pregnancy, her cooperation later will probably not be good. The chances of treating the infant under these conditions appear to be better if treatment is started early, especially with acetarsone as compared with arsenicals requiring injection. Against such routine treatment it may be argued that some healthy infants will be subjected to the possible complications of treatment, that the total expense of such a program would be greater, and that the results of therapy more difficult to evaluate, but these hardly, in our opinion, outweigh the benefit of early routine treatment.

CONCLUSIONS AND SUMMARY

Experience in our children's venereal clinics showed: (a) Frequent failure of adequate treatment of syphilitic mothers during pregnancy. Only 25 (13 per cent) of one group of 187 syphilitic women had been treated at all, and only 6 of these had received adequate treatment. (b) That about 71 (40 per cent) of a group of 180 parents did not bring their infants to the clinics, and of the 109 (60 per cent) who returned, only 58 (53 per cent) of these cooperated well. (c) That more than half of a group of 300 syphilitic children had not received treatment until they were five years of age or older.

There are many difficulties encountered in making an accurate diagnosis of syphilis in the first few weeks of life. In combination with the above factors, this means further delay in the institution of treatment.

Routine treatment of all infants born of syphilitic women who have not received adequate therapy during their pregnancy is an effective method of protecting infants from the disease. There are strong arguments in favor of such routine treatment, especially when the mother

is noncooperative and has been inadequately treated during pregnancy. In a series of 36 such infants, all were free from symptoms and all but one had a negative serologic reaction after one year of treatment.

Acetarsone compares favorably with other arsenicals in curing the lesions of syphilis and in producing negative serologic reactions.

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CONGENITAL SYPHILIS

II. Comparison of Treatment With Acetarsone and Other Arsenicals

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WE HAVE compared the results of treatment of syphilitic infants and children who received acetarsone as the only form of arsenic with those of patients who were treated with sulpharsphenamine and neoarsphenamine. In all groups the amount of treatment was considered to be adequate.

PROCEDURE

When the diagnosis of syphilis was definitely established, a course of acetarsone was instituted. At the beginning of the study, only infants under 2 years of age were treated, the dosage advocated by Maxwell and Glaser[1] being employed as follows:

0.05 gm. once a day for the first week
0.05 gm. twice a day for the second week
0.05 gm. three times a day for the third week
0.05 gm. four times a day for the fourth week
0.10 gm. three times a day for the fifth week
0.10 gm. four times a day for the sixth week
0.25 gm. twice a day for the seventh week

When attendance of the patient at the clinic was interrupted by minor respiratory infections or by contagious diseases, the dosage of acetarsone was not increased according to schedule, and occasionally it was reduced in amount depending upon the interval of absence. More recently, children from 2 to 15 years of age have been treated according to the same program. After each course of acetarsone therapy a rest period of one month was allowed, during which time the mother was instructed to give her child daily inunctions of 5 to 10 grains of mercurial ointment. One complete course of treatment, therefore, consisted of acetarsone administration for seven weeks and of mercury inunctions for four weeks.

The serologic tests employed were the Wassermann and Kahn reactions. A complete physical examination was performed at the beginning of the treatment, and the patient's symptoms and general condition were reviewed at each subsequent visit to the clinic, the urine was examined, and other tests such as a red blood count and hemoglobin estimation were performed when indicated.

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RESULTS

Only the patients who have been observed and treated for at least one year are included in this report. In the first group the 15 patients were from 3 to 23 months old and in the second group the 38 patients were from 2 to 15 years old when the treatment was instituted. The total series of 53 children received from 3 to 15 courses of treatment over a period of 12 to 54 months; an average of 7.5 courses were given during a period of approximately 26 months.

Results have been judged from the clinical findings and the blood serologic tests. Roentgenologic examination of the long bones and study of the cerebrospinal fluid have been made only when especially indicated. In Tables I and II the results have been classified according to the initial symptoms of the disease and in age groups above and below 2 years. All of these patients had positive blood serologic tests before the treatment was instituted. Two older children who were Wassermann fast, received a course of neoarsphenamine injections following the acetarsone therapy. To 1 child who did not tolerate acetarsone well was administered a series of ten bismuth injections after several courses of acetarsone. The trend of the disease was not changed in these 3 patients and they have been included in the series. Not included because of poor cooperation in regard to treatment was the case of 1 child, 4 years of age, who had a primary syphilitic chancre on the upper lip. In this case spirochetes were demonstrated in the lesion by dark-field examination. Treatment was started with acetarsone one month after the development of the chancre and continued for eight months. The lesion healed completely, and the reversal of a strongly positive Wassermann reaction to a negative one occurred in seven months. Observed again, after

TABLE I

SYPHILITIC INFANTS UNDER 2 YEARS OF AGE TREATED WITH ACETARSONE AND MERCURY

SYMPTOMS OF SYPHILIS	TOTAL NUMBER OF PATIENTS	RESULTS (AFTER 1 YR. OR MORE OF TREATMENT)		
		NEGATIVE SEROLOGIC TEST. NO ACTIVE LESIONS OR RESIDUAL SCARS	POSITIVE SEROLOGIC TEST. NO ACTIVE LESIONS OR RESIDUAL SCARS	POSITIVE SEROLOGIC TEST AND ACTIVE LESIONS OR SCARS
A. Active lesions (snuffles, skin rash, epiphysitis, etc.)	8	4	3	1
B. Chronic changes (enlarged lymph nodes, liver, spleen)	3	3	0	0
C. Latent—no symptoms or lesions	4	3	1	0
Total	15	10 (66.7%)	4 (26.7%)	1 (6.7%)

TABLE II

SYPHILITIC CHILDREN 2 YEARS TO 15 YEARS OF AGE TREATED WITH ACETARSONE AND MERCURY

SYMPTOMS OF SYPHILIS	TOTAL NUMBER OF PATIENTS	RESULTS		
		NEGATIVE SEROLOGIC TEST. NO ACTIVE LESIONS OR RESIDUAL SCARS	POSITIVE SEROLOGIC TEST. NO ACTIVE LESIONS OR RESIDUAL SCARS	POSITIVE SEROLOGIC TEST AND ACTIVE LESIONS OR RESIDUAL SCARS
A. Active lesions (keratitis, periostitis, flat condylomas, etc.)	8	1	4	3
B. Chronic lesions, (Hutchinson teeth, lymphadenopathy, etc.)	5	0	4	1
C. Latent — no symptoms or scars	25	12	13	0
Total	38	13 (34.2%)	21 (55.3%)	4 (10.5%)

a period of eighteen months, this patient was found to have remained serologically and clinically negative.

Period of Observation.—The children with serologic reversals have been observed for varying periods of time since these negative reactions were obtained. A few have been examined for as long as three or four years, but the majority have been under observation for less than the two years usually accepted as a minimum of time for the final judgment of effectiveness of "cures."

Complications.—The most frequent complications of the treatment were diarrhea and vomiting, especially in infants less than 2 years of age. Since these are common symptoms in infants at this age period, it was often difficult to determine whether acetarsone had been the sole cause. In at least 2 infants of the group of 15, however, the gastroenteric symptoms seemed to be definitely related to the ingestion of the arsenical. Diarrhea occurred in none of the 38 children 2 years of age or older, but the symptom of vomiting was noted twice. Albuminuria of a transient type occurred in 6 children over 2 years of age and in 1 of the infants. One of the older children had albuminuria and hematuria, and another had recurrent albuminuria, but both recovered completely. Other symptoms referable to arsenic or mercury therapy which developed in single instances were typical arsenical dermatitis, abdominal pain, and stomatitis—all in the older children.

In other children not included in the above series, disturbances of the gastroenteric system, skin rashes, and transient albuminuria have occurred with similar frequency. One severe case of aplastic anemia with complete recovery has been reported from this clinic.[2] After the administration of acetarsone for one week to an older boy an

interstitial keratitis developed which healed completely after intensive treatment with a bismuth compound, the acetarsone being continued.

Comparison of Acetarsone With Other Arsenicals.—We have attempted to compare the results obtained with acetarsone and with other arsenicals. The drugs used in the second group were sulpharsphenamine in infants and neoarsphenamine in older children. Various forms of mercury and bismuth were also employed. Six injections of an arsenical and ten of a heavy metal constituted one complete course of treatment in this group. All patients were proved to be syphilitic by serologic tests before treatment was started, and only those who had received at least one year of medication are included in the reported group. The majority of these patients were under observation during the same time as the acetarsone series. The results have been tabulated in Table III.

TABLE III

COMPARISON OF RESULTS WITH ACETARSONE AND WITH OTHER ARSENICALS

	NUMBER OF PATIENTS	AVERAGE NUMBER OF COURSES OF TREATMENT	AVERAGE TIME OF TREATMENT IN MONTHS	RESULTS		
				NEGATIVE SEROLOGIC TESTS. NO ACTIVE LESIONS OR RESIDUAL SCARS	POSITIVE SEROLOGIC TESTS. NO ACTIVE LESIONS OR RESIDUAL SCARS	POSITIVE SEROLOGIC TESTS AND ACTIVE LESIONS
Under 2 Years of Age						
Acetarsone	15	6.5	28	10 (66.7%)	4	1
Other arsenicals	19	5.2	36	15 (78.6%)	4	0
2 to 15 Years of Age						
Acetarsone	38	7.8	25	13 (34.2%)	21	4
Other arsenicals	62	7.3	42	13 (20.9%)	39	10

Since the numbers in each group were small, a statistical examination was made to evaluate (a) the results with acetarsone in the two age groups, (b) the differences between acetarsone and other arsenicals in children under 2 years of age, and (c) the differences of the 2 drugs in older children.

In each case "positive serology without active lesions or residual scars" and "positive serology with active lesions" were treated together as "positive." In comparing the age groups treated with acetarsone, there is obtained the figure $X^2 = 4.6121$. For this value of X^2, p is between 0.02 and 0.05, indicating that a difference in response as great as or greater than that observed between the two age groups would arise by chance alone somewhere between 2 and 5 times out of every 100 trials. In comparing acetarsone with other arsenicals there is obtained for the group under 2 years of age the figure $X^2 = 0.6495$. For this value of X^2, p is between 0.30 and 0.50. For the older group $X^2 = 2.1475$ and p is between 0.10 and 0.20.

If we accept values of p of 0.05 or less as indicating a significant deviation, we see that, in this series, the difference in response of the two age groups is a real one but that the differences observed in response to acetarsone and other arsenicals is a chance phenomenon.

COMMENT

A comprehensive review of the results of acetarsone therapy has been published recently by Whipple and Dunham.[3] There is a general impression that the drug is effective in curing the majority of syphilitic lesions and, in addition, that it has a general tonic effect and improves the nutritional state of the patients. The special advantage of the drug is its ease of administration and the consequent improvement of cooperation of the parents of syphilitic children. Our experience leads us to agree with these conclusions and also to believe that acetarsone compares favorably with other arsenicals.

The major problem in the treatment of congenital syphilis with acetarsone or any other drug is early and continuous therapy. In a series of 300 syphilitic children who have attended our clinics, the disease manifested active symptoms in one-third; the remainder were diagnosed by positive serologic tests taken only because of a history of the disease in the family or by routine serologic tests when the patients were ill from other diseases.[4] It must be, therefore, that a great number of syphilitic children never come under observation until the severe manifestations of the disease develop in later years of childhood.

It is difficult to obtain cooperation of the majority of the parents of patients for an adequate length of time to insure optimum benefits from any form of treatment. The above series of 134 patients who have received a year's treatment represent only a small percentage of the entire number who have attended the clinics, the remainder coming irregularly or for short periods of time only.

The advantages of treatment of congenital syphilis with any type of arsenical are so much greater in the early months of life that the important phase of this subject would seem to be the time of administration rather than the nature of the arsenical employed.

SUMMARY

Acetarsone and mercury inunctions were administered to a group of 53 children for periods of twelve to fifty-four months. These patients received an average of 7.5 courses over an average of a twenty-six-month period.

The results are statistically much better in younger children. In 10 of 15 (66.7 per cent) under 2 years of age the serologic tests were negative after one year of treatment; in 4 (26.7 per cent) the tests

were positive, but there were no symptoms; in 1 (6.7 per cent) the test was positive and active lesions persisted. In only 13 (34.2 per cent) of 38 children between 2 and 15 years of age were the serologic tests negative after one year of treatment; in 21 (55.3 per cent) the serologic tests were positive, but symptoms or signs were absent, and in 4 (10.5 per cent), both positive serologic tests and symptoms or scars were present.

The treatment with acetarsone has been compared with other forms of arsenic. Negative serologic tests and freedom from symptoms were obtained in approximately 67 per cent of 15 infants less than 2 years of age with acetarsone and in 79 per cent of 19 infants treated with sulpharsphenamine and neoarsphenamine. In older age groups, 34 per cent made good progress with acetarsone and 21 per cent with other arsenicals. These differences do not appear to be statistically significant.

The most common complications of the acetarsone treatment were digestive disturbances, albuminuria, and skin rashes, which occurred in all age groups and were transitory in nature.

We wish to express our gratitude to Mrs. W. E. Brown for her assistance in preparing the statistical analysis, and to the resident physicians of the Pediatric Department who have operated the venereal disease clinics during the past seven years.

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THE SUPPLEMENTARY VALUE OF THE BANANA IN INSTITUTION DIETS

I. Effect on Growth in Height and Weight, Ossification of Carpals, and Changes in Franzen Indices

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IT WAS pointed out in an earlier article[1] that one approach to what constitutes an adequate diet in childhood is through supplementary feeding experiments in which various types of supplements are added to the diets of children under controlled conditions and the effect on growth and other measures of physical status is noted. According to the theory advanced by Orr,[2] if any significant improvement results, it indicates that the previous diet was below optimum in respect to some of the dietary constituents contained in the supplement. If this theory be accepted, then a series of such studies should help materially in arriving at standards for optimal intakes of the various dietary essentials.

The present study is of this type. It reports the results of supplementing the diet of over 100 boys living in one institution with two to three bananas daily over the period of the school year. Informal observations indicated that the diet of these boys was probably reasonably adequate, for it contained a quart of milk per boy daily as well as fair amounts of other basic foods, and the weights of the boys were mostly within the "normal" range. It was the judgment of the observers that the diet was most questionable in respect to vitamin C and total calories. When therefore the opportunity was afforded to test the value of the banana as a supplementary food, it was welcomed as a means of checking these conclusions for this institution, and making thereby a contribution to the knowledge of nutritional requirements of children. The banana was a highly suitable food to use for this purpose from several standpoints: (1) It is a palatable food, almost universally liked by children; (2) it is easily administered since it requires no preparation, no dishes or utensils for serving, and, hence, no dishwashing afterwards; and (3) it supplies both calories and vitamin C as well as other dietary constituents in significant amounts.

EXPERIMENTAL

Subjects.—The subjects of this study were boys ranging from 8 to 16 years of age, living in an institution. The boys live in cottages, about 30 to a cottage, each under the care of a matron, but all eat in

*From the Department of Home Economics, University of Chicago. The expenses of this study were defrayed in part by the United Fruit Company.

a common dining hall. Eight cottages of the younger boys who were still in the period of rapid growth were selected for the study. This included a total of 277 boys, 123 in the experimental and 154 in the control cottages. Excluding those who were absent for any considerable period or who were transferred from one group to the other during the study, there remained 96 experimental and 101 control boys to be used for the final analysis. For most evaluations, groups matched on the basis of pertinent factors were chosen from these larger groups, as will be described later.

General Plan of the Study.—The study continued over the school year of nine months, from October to June. It had been planned to cover the full calendar year, but, since most of the boys are away for some weeks during the summer, it seemed wiser to restrict it to the school period. At the beginning of the year a variety of measures of physical status were applied to all boys in the eight cottages. Following this, the supplement was given to the boys in the four experimental cottages while the other four remained as controls. At the end of the nine months, the same measures were again applied, and the progress of the two groups was compared. Certain measures as specified below were repeated at more frequent intervals.

Administering the Bananas.—Since it was intended that the bananas be eaten *in addition to* the regular diet, and since not all boys were to receive them, they were given by the matrons in the cottages at times when they would be least likely to interfere with the appetite for the regular meals, namely, one after school in the afternoon and the others after supper in the evening. The worker in charge of the project supervised the feeding of the bananas during the first few days until she was sure that the matron could be trusted to carry it on and to keep accurate records. There was never any question as to the dependability of the matrons and the strictness of their procedure. The bananas were delivered in boxes twice weekly to each of the cottages and kept in a locked storeroom to which only the matron had access. At the time for serving they were brought out, dispensed by the matron, and eaten in her presence. The boys were allowed as many as they wanted up to three in the older groups and two in the younger ones, and, as will be shown later, most boys took full advantage of their opportunity. The number eaten by each boy was recorded by the matron in a table provided for this purpose.

Measures of Physical Status.—All available measures of physical status which might be related to the supplement were utilized to assess the relative progress of the two groups. These included (1) measurements of height and weight taken at four-week intervals; (2) roentgenograms of the wrists taken at the beginning and end of the study; (3) measurements for the Franzen indices, also at beginning and end; (4) the capillary resistance test determined four times during the year;

and (5) the reduced ascorbic acid in the blood plasma at the end of the experiment. The first three of these measures were employed as criteria of general growth and development, the last two as indices of the state of nutrition with respect to vitamin C. A quantitative dietary study was also made at the beginning of the study to determine the amounts of the various constituents in the diet to which the supplement was to be added. A record of colds and other illnesses and indispositions was also kept to be used in interpreting the results.

TABLE I

CONSUMPTION OF BANANAS BY EXPERIMENTAL BOYS

NO. OF BANANAS	BY ALL 96 BOYS		BY 80 MATCHED FOR SIZE	
	NUMBER	PER CENT	NUMBER	PER CENT
Total consumed by all	42,741*		36,723	
Individual consumption				
Average	445		459	
600 or more	8	8	8	10
500-599	27	28	23	29
400-499	23	24	23	29
300-399	28	29	19	24
200-299	10	10	7	8

*The total actually eaten in the institution was 48,865 bananas; the 6,214 additional ones were eaten by boys dropped from final analysis.

TABLE II

NUMBER AND PERCENTAGE OF CHILDREN CONSUMING SPECIFIED PERCENTAGE OF BANANAS AVAILABLE*

PERCENTAGE OF AVAILABLE BANANAS	ALL 96 BOYS		80 MATCHED FOR AGE		
	NUMBER	PER CENT	NUMBER	PER CENT	
95-100	41	43	37	46	80
90- 95	13	14	12	15	
85- 90	17	18	15	19	
80- 85	7	7	5	6	
75- 80	7	7	3	4	
Less than 75	10	10	8	10	

*The high degree of acceptance is also shown by the fact that 92 per cent of all bananas available were consumed.

Consumption of Bananas.—The total and per capita consumption of bananas is given in Tables I and II. During the nine months approximately 50,000 bananas were provided the institution. For the 96 boys who remained to the end of the study, the total consumption was 42,741 bananas, or an average of 445 per boy, and for a matched group of 80 boys who were used for most of the comparisons, the average was 459 per boy. The number eaten by individual boys varied greatly. Thirty-six per cent of the boys ate 500 or more; 8 per cent of these consumed 600 bananas during the period. Only 10 per cent of the group ate fewer than 300 bananas, and these were mostly the smaller boys. The bananas were well liked at all times and were consumed with the same eagerness and dispatch at the end of the nine months as at the beginning. A large proportion of the boys regularly consumed their full allowance; that is,

three or two according to the age. As shown in Table II, 80 per cent of the boys in the matched group ate 85 per cent or more of the bananas available, and 46 per cent of them consumed 95 to 100 per cent of their quota.*

To afford some tangible idea of the supplementary value of bananas eaten in these amounts, the quantities of the various dietary essentials provided by the average of 459 bananas per boy consumed during the study are shown in Table III, together with the amounts of these constituents obtained by boys who regularly ate two or three bananas daily throughout the study. It is apparent that these amounts provided substantial additions of several dietary constituents, notably 200 to 300 calories and 340 to 510 international units of vitamin C daily for a nine-month period. The significance of these additions will be discussed in the next section.

TABLE III

FOOD CONSTITUENTS PROVIDED BY BANANA SUPPLEMENT

	NUMBER BANANAS	CALORIES	PROTEIN (GM.)	CALCIUM (GM.)	PHOSPHORUS (GM.)	IRON (MG.)	VITAMINS			
							A (SHERMAN UNITS)	B_1 (INTERNATIONAL UNITS)	C (INTERNATIONAL UNITS)	G (RIBOFLAVIN) (SHERMAN UNITS)
Average total* for each of 80 boys for year	459	45,900	606	3.7	13.8	289.0	109,242	6656	78,030	13,770
Daily average for boys having 2 per day	2	200	2.6	0.016	0.06	1.3	476	29	340	60
Daily average for boys having 3 per day	3	300	4.0	0.024	0.09	1.9	714	44	510	90

*The average number of days in which bananas were eaten was 223. The daily average per boy would thus be 2.1 bananas.

The Nature of the Diet to Which the Supplement Was Added.—In interpreting the findings of the study, it is essential to know with as much exactness as possible what the diet of the boys provided in the way of dietary constituents, and how these were affected by the banana supplement. A quantitative dietary study was carried out at the beginning of the year. It covered a two-week period and included 113 boys about equally divided between control and experimental cottages. The details of the method were the same as commonly used in this laboratory.[3, 4] Using the data so obtained, the calorie and protein intake for each boy and the mean for each age group were calculated. The

*The acceptance was, in fact, probably much higher than this, for the failure to have the after-school banana was frequently due to the boys' staying after school for baseball practice and arriving home too late to receive it. The records showed only whether or not a boy ate the bananas, not the reason for his failure to do so, except when he was absent from the institution or in the hospital.

TABLE IV

MEAN INTAKE OF DIETARY CONSTITUENTS FOR BOYS 10 TO 12 YEARS OF AGE AND EFFECT OF BANANA SUPPLEMENT*

	CALORIES	PROTEIN	CALCIUM	PHOS.	IRON (MG.)	VITAMINS†			
						A S.U.	B (THIAMIN) (I.U.)	C (ASCORBIC ACID) (I.U.)	G (RIBO-FLAVIN) (S.U.)
Mean intake	2624	81.0	1.33	1.67	13.0	4977	299	580 (480)	769
Total with two bananas	2824	82.6	1.35	1.73	14.3	5453	328	920 (820)	829
Percentage increase	7.6	4.0	1.5	3.50	10.0	10.0	10.0	60	8
Standards									
Steibeling‡	2400-3000	75.0	0.88-1.0	1.2-1.3	12-15	3500-4000		900-1000	
Range of other authorities	2100-4000	50-100	1.00 all ages	1.0-1.5	10-18	3000-8000	150-250	600-2000	400-700

*Calories and protein are results of two-week quantitative dietary study of 96 boys of these ages. Minerals and vitamins computed from diets of boys whose intakes were typical of the average.

†Vitamin values computed from Daniel and Munsell. Vitamin Content of Foods, U. S. Dept. of Agri. Bull. No. 275, 1937. Vitamins B, C, are given in international units, A and G in Sherman units.

Reduction of about 50 per cent in B and of 50 to 66 per cent in vitamin C was made for long-cooked foods. It is probable that the vitamin C suffered even greater destruction. If so, the values would be reduced to those shown in parentheses. A few foods for which no figures were available were omitted.

‡H. K. Steibeling and M. M. Ward: Diets at Four Levels of Nutritive Content and Cost, U. S. Dept. Agri. Circular No. 296, 1933.

complete results of this portion of the study will be reported in another connection. For the present purpose diets representative of the average intakes of boys of the different age groups were utilized to compute the minerals and vitamins contributed by the institution diet. The mean intake so obtained for boys 10 to 12 years of age is given in Table IV, together with some of the dietary standards which have been suggested by various authorities for the several dietary essentials. The contribution to this diet made by the banana supplement is also included.

The results bear out the prediction of the writers that the diet would measure well up to the usual standards in most respects. The standards for all constituents vary greatly as would be expected since most of them are based on very little experimental data. If the mean intakes in the table are compared with the values suggested, it will be seen that the diet of this institution measures well up to all standards for calcium, phosphorus, and vitamins A, B_1, and G; it meets the minimum but not the optimum standards for calories, protein, iron, and vitamin A, and it is decidedly questionable in vitamin C. In vitamin C the intake as calculated is slightly below even the 600 international units commonly regarded as a minimum standard. The intake may moreover have been actually lower than this, for even more of the vitamin in cooked foods may have been destroyed than was allowed for in the calculations. If the questionable amounts in the long-cooked vegetables be disregarded and only the more certain amounts derived from the quart of raw milk, potatoes, and the small amount of raw cabbage and apples be counted, the average intake would be only 430 units. Though these values are probably representative of the average intake in October, they are not of course accurate for individual boys, nor for the intake at other seasons. The boys ate varying amounts of the vitamin C-containing foods, especially potatoes, and the supplementary fruit occasionally brought by relatives varied with individuals. Even the small amounts of fresh fruits and vegetables were decreased in the fall, winter, and early spring months, with a consequent reduction in vitamin C. In the last few weeks of the study, rhubarb and a few early vegetables were coming on in the garden, and these probably increased the intake somewhat during this time, especially for the boys who worked a few hours a day in the farm garden and who helped themselves to such as were available.

It is of interest now to note additions the banana made to this diet. In Table IV the constituents contributed by two bananas have been added to the mean intake, and the percentage increase in each item determined. It is observed that the increase is of practical significance for all items except protein, calcium, and phosphorus. The increase amounts to 8 per cent in calories and in vitamin G, and 10 per

cent in iron and in vitamins A and B_1. The most significant contribution is a 60 per cent increase in vitamin C, the constituent of the diet which most needed supplementing.

EFFECT OF THE SUPPLEMENT

It is now pertinent to inquire whether these additions effected any significant improvement in the physical status of the children receiving them as measured by their progress in comparison with that of the control group.

Growth in Height and Weight.—Cognizant of the many pitfalls inherent in the usual methods of analysis of data from this type of group study, every effort was made to avoid them as far as possible through a variety of approaches to the problem, and particularly through the use of carefully matched groups. For the appraisal of growth, two groups were formed by matching pairs from control and experimental houses on the basis of age, height, weight, percentage deviation from expected weight for height and age, and the gain in weight to be expected for the period. It was impossible, of course, to match them exactly on all bases, but by giving the odds on the different items first to one group then to the other, two groups consisting of 80 such pairs were formed which averaged essentially alike on the bases indicated. This is shown in Table V, in which the means for the two groups at the beginning are recorded. The average age differs by less than 0.1 year, the weight by only 0.5 pound, and other items differ in no greater degree. The close similarity of these means should not, however, blind us to the fact that they are averages only, that individual pairs within the groups differ considerably, and that "expected weight" and "expected gain in weight" are theoretical assumptions which may not be entirely accurate for any individual boy in question. Nevertheless, such discrepancies as do occur are distributed impartially to both groups, and there can be no doubt that the use of such matched groups constitutes a sounder procedure than does the method commonly employed of using unselected groups varying in number, size, and age of subjects.

TABLE V

MEAN STATUS OF TWO GROUPS OF 80 BOYS EACH MATCHED FOR AGE, HEIGHT, AND WEIGHT AT THE BEGINNING OF THE STUDY

GROUP	MEAN AGE (YR.)	MEAN HEIGHT (IN.)	MEAN WEIGHT (LB.)	MEAN PERCENTAGE DEVIATION FROM EXPECTED WEIGHT	MEAN GAIN EXPECTED DURING PERIOD (LB.)
Control	12.4	58.2	84.9	+0.7	4.7
Experimental	12.5	58.2	85.4	+1.1	4.6

The progress of the two groups in height and weight during the experimental period has been assessed on three bases: (1) mean gains

made by the two groups; (2) percentage of expected gain made by the two groups; and (3) relative gains of matched partners. The results of these analyses are shown in Tables VI and VII and in Chart 1. It is evident by all three methods of evaluation that the odds are consistently in favor of the experimental group. The actual difference in mean gains is small, amounting to 0.90 pound in weight and to 0.15 inch in height (Chart 1, *A*). These differences do not quite attain statistical significance, being respectively only 2.2 and 3.4 times their probable errors. Their significance is, however, enhanced by the

TABLE VI

MEAN GAIN IN WEIGHT AND HEIGHT OF MATCHED GROUP OF 80 CONTROL AND 80 EXPERIMENTAL BOYS

	CONTROL	EXPERIMENTAL	DIFFERENCE WITH P.E.	RATIO OF DIFFERENCE/P.E.
Mean gain in weight (pounds)	6.0	6.9	0.90 ± 0.4	2.2
Mean gain in height (inches)	1.38	1.53	0.15 ± 0.046	3.2
Mean percentage of expected gain in weight made by group	105.0	128	28 ± 6.7	3.4
Mean percentage of expected gain in height made by group	105.0	127	22 ± 4.5	5.0

TABLE VII

PER CENT OF 80 MATCHED CHILDREN IN EACH GROUP MAKING SPECIFIED PERCENTAGE OF EXPECTED GAIN IN HEIGHT AND WEIGHT

PER CENT OF EXPECTED GAIN	WEIGHT				HEIGHT			
	CONTROL		EXPER.		CONTROL		EXPER.	
	NO.	%	NO.	%	NO.	%	NO.	%
Total	80		80		80		80	
100 per cent or better	35	43.8	56	70	46	57.5	57	71.2
200 and more	9		12		3		9	
150 to 199	5		16		6		13	
100 to 149	21		28		37		35	
Less than 100 per cent	45	56.2	25	30	34	42.5	23	28.8
50 to 99	27		17		31		23	
0 to 49	18		8		3			
Loss	3		1					

fact that they are based on matched groups, not merely random samples of the population. A fairer method of comparing progress, however, is on the basis of expected gains, since each child is thus judged by his own standard. By this method the differences are more pronounced (Tables VI and VII, Chart 1, *B*). The percentage of individual boys who made their expected gains in weight or better is 70 for the experimental as against 44 for the control group; for height the percentages are 71 and 58 for the two groups, respectively. When each group is considered as a unit the mean percentage gain is found to be 127 for weight and 128 for height for the experimental boys, as com-

pared with a mean percentage gain of 105 for both weight and height for the control boys (Chart 1, *C*). The differences between these means for the two groups are, moreover, probably significant as they are 3.4 (for weight) and 5 (for height) times their probable errors.

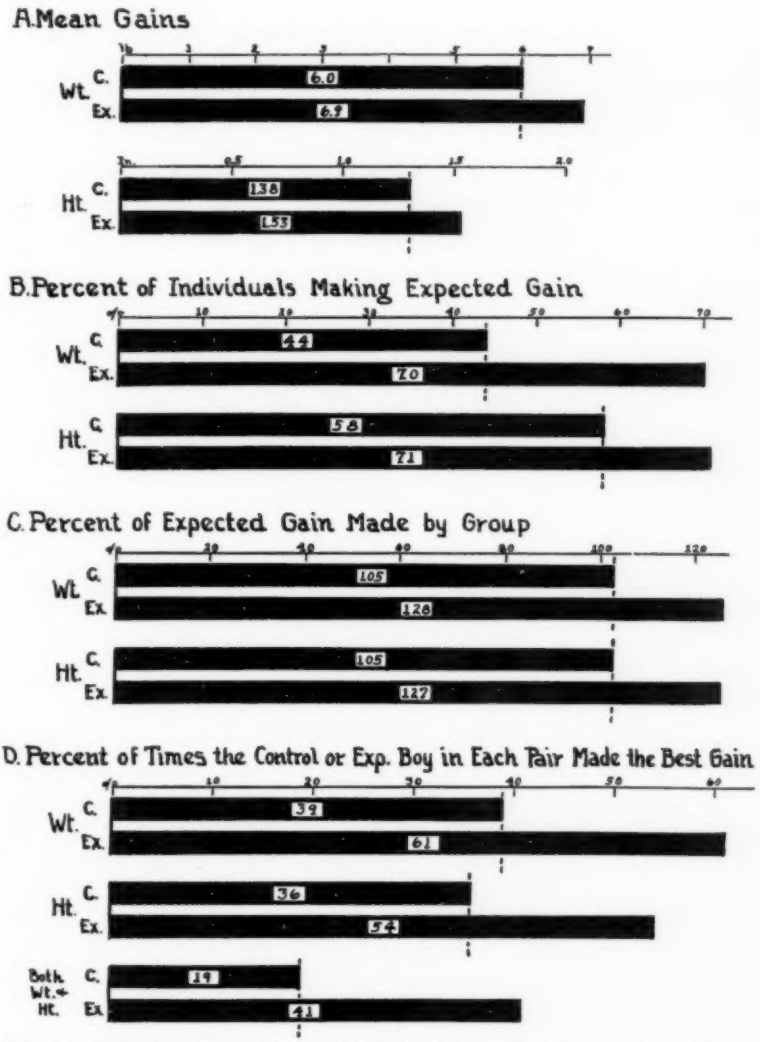

Chart 1.—Comparison of growth in weight and height of control and experimental boys.

When individuals in two groups are paired as closely as possible at the outset, it is always of interest to note the relative progress of the partners in each of the pairs. It would not be expected that the

experimental boy should always exceed his control partner, for other uncontrolled variables of necessity enter in and the partners cannot be perfectly matched. Nevertheless, since the matching was fairly well done and the supplement the only variable applicable to all, the odds should be on the side of the experimental group even by this severe test. Such was the result, moreover, in the present study (Table VIII, Chart 1, *D*). Out of the 80 comparisons for weight, the experimental partner made the best gain in 61 per cent, the control in 39 per cent; in height, best gains were made by 54 and 36 per cent of the respective partners. Moreover, best gains in both height and weight (or best in one and equal in the other) were made by the experimental boys in 41 per cent, by the control in only 19 per cent of the comparisons; in the remainder, the honors were equally divided.

TABLE VIII

TIMES EXPERIMENTAL AND CONTROL PARTNERS IN THE 80 PAIRS MADE BEST GAIN IN HEIGHT, WEIGHT, AND IN BOTH

	NUMBER	PER CENT
TOTAL	80	100
Best weight gain by:		
Experimental partner	49	61
Control partner	31	39
Best height gain by:		
Experimental partner	43	54
Control partner	29	36
Groups even	8	10
Best gain in both height and weight (or best in one, even in other):		
Experimental partner	33	41
Control partner	15	19
Height best in one, weight in other	32	40

On all comparisons of growth in height and weight, then, the boys receiving the supplement had some advantage; on the basis of percentage of expected gains made by the respective groups these differences are statistically significant. Since the groups were well-matched at the outset and since there was no other known variable that did not affect both groups equally, it seems fair to conclude that the supplement did induce slightly greater gains in the experimental boys.

*Gains in Ossification of the Carpals.**—As already indicated, roentgenograms of the wrists of the boys in both groups were taken at the beginning and again at the end of the study in order that the progress in ossification of the two groups might be compared. The method of taking the roentgenograms and for measuring the ossified area in the films has been described fully in a previous article.[5] The essentials of procedure necessary for interpretation of the data here presented may be briefly stated:

*The x-ray pictures for this portion of the study were taken by Vera MacNair. The films were measured and the matchings and calculations made by Grace Steininger.

The measurement of the degree of ossification was made by the method devised by Freeman and Carter:[6] The shadows of the ossified areas of the carpals and epiphyses as shown in the films are measured by means of a planimeter; the sum of these constitutes the total area ossified. A quadrilateral which represents the total size of the wrist area is similarly measured; this constitutes the area to be ossified. The sum of the ossified areas of carpals divided by the wrist area gives the ossification ratio, that is, the ratio between the area *already ossified* and the area *to be ossified*. These ratios constitute a series of values for the stage of ossification that may be compared for individuals or groups in much the same way as weight and height are. From tables of age norms prepared by Freeman and Carter and extended by Flory, the expected ossification ratio of any given child may be obtained, and his percentage deviation from the norm computed. Furthermore, if his ossification ratio is determined at the beginning and end of a period of time, as in the present study, the difference between the two ratios will represent his gain during the period. By reference to Carter's tables the expected gain during the period may be obtained, and his actual gains may be expressed in per cent of the expected just as is done for height and weight. Moreover, by totalling actual and expected gains of all individuals in a group, the gain of each group as a whole, as well as the mean gain, may also be determined; this, too, may then be expressed as the percentage of the expected. Such computations were made in the present study for both experimental and control groups.

How did the two groups compare in their progress in ossification? Since the boys differed at the outset in age, size, and in stage of ossification, it was deemed essential that comparisons be made on groups matched for these items. Accordingly, the initial films of boys from control and experimental groups were paired as closely as possible for age, height, Flory age index,[7] Carter ossification ratio, and gain in the Carter ratio to be expected during the period. There were 53 pairs who could be reasonably well matched on these bases. Small differences between individual partners, moreover, cancel each other when each group is considered as a unit, so that the mean status of experimental and control groups is found to be almost identical. This is shown in Table IX.

TABLE IX

MEAN STATUS OF TWO GROUPS MATCHED FOR AGE, OSSIFICATION AND EXPECTED GAIN AT THE BEGINNING

	NUMBER	MEAN AGE	MEAN CARTER RATIO	MEAN PERCENTAGE DEVIATION FROM AGE STANDARD	MEAN GAIN IN CARTER POINTS EXPECTED	MEAN FLORY AGE IN MONTHS
Control	53	12.1	81.53	6.06	3.89	143.3
Experimental	53	12.1	81.36	6.14	3.89	143.3

The mean age is 12.1 years for each of the groups, and the means for the other items are either precisely alike or differ by insignificant amounts. The two groups can, therefore, be considered as fairly well matched at the outset and suitable for determining differences in progress. The comparisons made were similar to those for weight and height, namely, (1) mean gains in Carter ratio; (2) percentage of expected gain in Carter ratio; (3) percentage of individuals in each group making expected gains; and (4) relative gains of partners in the matched pairs. The results of these evaluations are given in Table X and Chart 2.

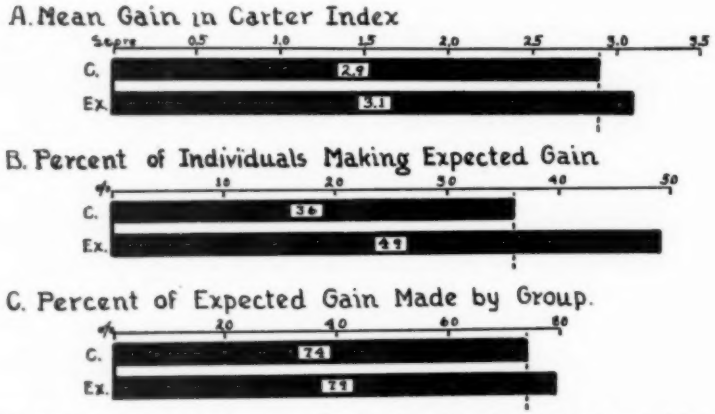

Chart 2.—Comparison of gains in ossification of wrists of control and experimental boys, expressed by the Carter index.

It will be observed that the differences in mean progress made by the groups are small in all comparisons, but those that do exist are in favor of the experimental boys. When mean gain in ossification ratio is considered (Chart 2, *A*), the difference is insignificant, 3.1 versus 2.9 Carter points. On the basis of percentage of expected gains made, which is the fairest method of appraisal, the odds for the experimental boys are more apparent (Chart 2, *B*). Forty-nine per cent of the ex-

TABLE X

OSSIFICATION RATIO COMPARISON OF PROGRESS DURING EXPERIMENTAL PERIOD OF TWO MATCHED GROUPS

GROUP	NUMBER	MEAN GAIN IN CARTER POINTS	PER CENT OF EXPECTED GAIN MADE BY GROUP	PER CENT OF INDIVIDUALS MAKING EXPECTED GAIN OR BETTER	PER CENT MAKING GAINS OF 200% OR MORE	TIMES GAINS EXCEED THOSE OF PARTNERS	
						NUMBER	PER CENT
Control	53	2.9	74.3	35.8	11	22	41
Experimental	53	3.1	79.2	49.1	17	27 (4 tied)	51

perimental boys made their expected gain in Carter index, as against 36 per cent of the controls, and the percentages making large gains of 200 per cent or more of the expected are 17 and 11 for the same groups respectively. Considering each group as a unit (Chart 3, *C*) the experimental boys made 79 per cent, and the controls, 74 per cent of their expected quotas. Even on the basis of relative gains of matched partners the experimental boys have a slight edge. In 51 per cent of the matched pairs the experimental boy made a greater gain in Carter index during the year than his control partner, as against 41 per cent in which the control boy had the advantage; in the remaining 8 per cent the gains for the partners were equal.

It was not expected that any marked difference in ossification in the two groups would be observed over the period studied, which for this item amounted to but little over six months. The diet, moreover, was well fortified with calcium and phosphorus, the chief bone-building materials, and may also have been adequate in the vitamins needed in their utilization. Nevertheless, since the diet appeared to be suboptimal in vitamin C, and since this vitamin is believed to be related to calcium metabolism, it seemed pertinent to determine whether any difference in favor of the supplemented group could be observed. As has been noted, the differences found were slight and statistically insignificant. Their chief claim to recognition comes from the consistency of their direction and the fact that the groups were so closely matched at the outset. A longer period, however, would be required to determine whether or not this difference is a real one attributable to the supplement.

Changes in Franzen Measurements.—As another approach to the change in nutritional status of the two groups during the period, comparison was made of their progress by the Franzen indices of nutrition. Using the Franzen technique and instruments, measurements were made of arm girth, subcutaneous tissue, height, hip width, chest depth, and chest width at the beginning and end of the year.[8, 9] Scores for arm girth, subcutaneous tissue and weight as used by Franzen were also computed both for initial and final measurements. It will be recalled that Franzen's method attempts to assess the amount of soft tissues (muscle and fat), in relation to the size of the skeleton. These relationships are expressed as scores. The scores are derived by a series of computations in which the actual measurements of the subjects for arm girth, subcutaneous tissue, and weight are reduced to indices that make allowance for the skeletal build as indicated by hip, chest, and height measurements. This computation is facilitated by the use of tables prepared for this purpose.[9] The score for a given subject for any item having been derived, it is possible to determine how he compares with the general population by reference to a percentile table in which is shown the number of children in each 1,000 who might be expected to have scores below each specified level. Thus, for example, if a boy's arm

girth score is 35, reference to the table shows that his rank is 67. This means that only 67 boys out of every 1,000 have scores lower than his. He is, therefore, much below average in arm girth in relation to the size of his skeleton. In similar manner his rank in subcutaneous tissue and weight indices can be determined. In using this method to select undernourished children, Franzen has suggested that subjects having scores of 46 or lower on all three items—arm girth, subcutaneous tissue, weight—should be regarded as undernourished.

Space does not permit any further description of the details of the Franzen method nor any discussion of its validity or usefulness as a measure of nutritional status. These must be reserved for later presentation. For the present purpose the measures are merely accepted as indices that may be applied impartially to both dietary groups as additional indications of their relative progress. No suggestion has been made by Franzen as to how they might best be used for this purpose. After due consideration of all possibilities, it was decided to subject the data to the same types of analysis used for growth. Comparisons were thus made for mean gains in actual arm girths, subcutaneous tissue, and weight, and also for mean gains in the scores for these same items. Analyses were made for all boys for whom beginning and final measurements were made and who had been in their respective dietary groups throughout the study. Since the Franzen tables do not extend beyond 12 years, the older boys in this study were necessarily excluded. The total number suitable for use was 113 boys about equally divided between the two groups.

TABLE XI

CHANGES IN FRANZEN MEASUREMENTS OF GROUPS MATCHED AT OUTSET ON SPECIFIED ITEM

BASIS OF MATCHING	NO. PAIRS	MEAN FOR GROUP AT BEGINNING		MEAN GAIN		DIF. AND P.E.	RATIO OF DIF. / P.E.
		CONTROL	EXPER.	CONTROL	EXPER.		
Arm girth, cm.*	45	48.6	48.6	0.7	1.2	0.5±0.16	3.1
Subcutaneous tissue, mm.	55	24.8	24.8	-1.5	-0.6	-0.9±0.26	3.4
Weight (pounds)	44	75.4	75.4	+5.6	+6.6	1.0±0.47	2.1
Arm girth score	47	58.7	58.7	0.9	1.0	0.1	--
Subcutaneous tissue score	48	50.2	50.2	-3.9	-2.4	-1.5±0.84	1.8
Weight score	50	50.8	50.8	2.1	3.9	1.8±1.31	1.4

*Sum of flexed and relaxed arm measurements.

As in previous sections, it was decided that the fairest comparisons would be those based on groups matched at the outset for the item in question. Accordingly, boys from control and experimental groups were matched for initial arm girth, and the two groups of 45 pairs each thus obtained used to measure the relative progress on this

item during the year. They were then rematched on the basis of subcutaneous tissue and again successively for weight and for each of the three scores. In each case the matched groups were used to measure the year's progress on the item on which they were matched. The boys included in the various matchings were, of course, largely, though not entirely, the same, since they were derived from the same larger groups, and in each matching only a few extremes for whom no suitable partners in the other group could be found for the item in question were excluded. As seen in Table XI, the number of pairs in the various matchings ranges from 45 for arm girth to 55 for subcutaneous tissue. The closeness of the agreement between pairs is shown by the fact that in each of these matchings the means for the two groups at the beginning are identical, the small differences that existed between individuals having cancelled each other in the total group. With groups thus matched at the outset, it seems more probable that differences between them at the end of the year may be attributed to the dietary regime.

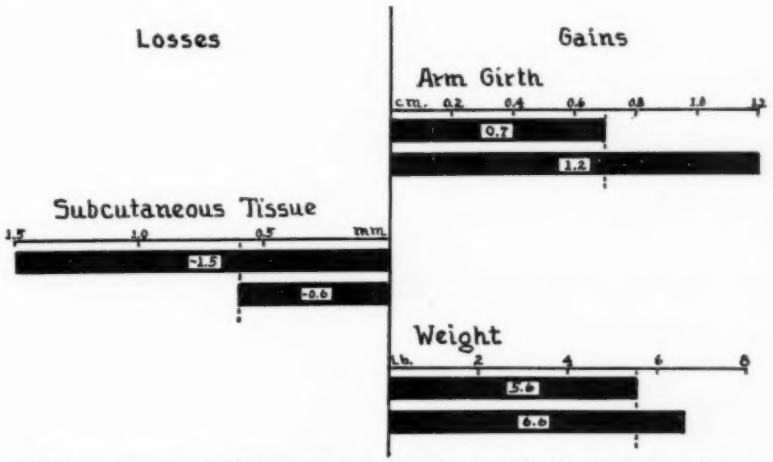

Chart 3.—Changes in Franzen measurements for control and experimental boys.

As may be seen in Table XI and Chart 3, both groups showed a mean gain in actual arm girth, in weight, and also in the scores for these two items for the period studied, while for the subcutaneous tissue measurement and the score for this item there was a loss, though that of the experimental group was less than that of the controls. This would seem to indicate that the gain in weight was due to growth in bone and muscle while there was an actual decrease in the layer of subcutaneous fat.

When the mean progress of the two groups is compared on these various bases, the differences are found to be small for every item of

the test, but, as in all previous evaluations, they are in all cases in favor of the experimental boys. The differences in mean gains for the two groups in actual measurements of arm girth, subcutaneous tissue, and weight, moreover, approach significance, being 3.4, 3.1, and 2.1, respectively, times their probable errors. For the three scores for these items, the differences are in the same direction but insignificant. It may be thus concluded that the boys receiving the supplement showed a slightly greater gain in arm girth and weight and a smaller loss in subcutaneous tissue during the year than did the control boys. This difference was evidenced both on the basis of actual measurements of these items, and on the scores which relate them to the skeletal build.

It is pertinent now to inquire how many boys in this institution would be "selected" as undernourished by the Franzen method, and whether this number changes during the year. The answer to this question is found in Table XII. If the basis of selection is a score of 46 or below on all three items (arm girth, subcutaneous tissue, and weight) as Franzen suggests, 5 boys in the experimental group and 4 in the control group would be considered as undernourished at the beginning, and 3 and 4, respectively, at the end. Although not suggested by the author of the method, it occurred to the writers that low scores on any two items might well be regarded as a basis for selection. On this basis the largest proportion is "selected" by the combination of weight plus subcutaneous tissue scores; 18 per cent of the experimental boys and 16 per cent of the control boys have scores of 46 or

TABLE XII

NUMBER AND PERCENTAGE OF BOYS IN CONTROL AND EXPERIMENTAL GROUPS WHO WOULD BE SELECTED AS BELOW NORMAL BY THE FRANZEN MEASUREMENTS BY VARIOUS METHODS AT BEGINNING AND END OF STUDY

ITEM	CONTROL				EXPERIMENTAL				CHANGE IN PER CENT SELECTED AT THE END*	
	BEGIN.		END.		BEGIN.		END			
	NO.	%	NO.	%	NO.	%	NO.	%	EXPERIMENTAL	CONTROL
A.G. score†	8	15	7	13	7	13	7	13	-2	0
S.T. score‡	19	35	24	44	18	33	30	55	+9	+20
Wt. score	16	29	9	16	20	36	15	27	-13	-9
A.G. and S.T. scores	6	11	5	9	5	9	6	11	-2	+2
A.G. and Wt. scores	5	9	4	7	5	9	5	9	-2	0
Wt. and S.T. scores	10	18	6	11	9	16	11	20	-7	+4
A.G., S.T., and Wt. scores	5	9	3	6	4	7	4	7	-3	0

*These figures represent the difference between the percentage selected at the beginning and at the end. A "-" sign then means improvement, since fewer are selected, and a "+" sign a less favorable status.

†A.G. = Arm girth.

‡S.T. = Subcutaneous tissue.

below on both these items at the beginning, and 11 and 20 per cent, respectively, at the end. If low scores on a single item are considered as indicating inferior nutrition, the largest proportion falls below on the subcutaneous tissue score, and the least on arm girth.

There is no marked change in the status of either group during the year (Table XI), but again the experimental boys show some advantage. This is best observed in the last column of Table XII, in which the change in the percentage "selected" is recorded. A minus sign signifies improvement since it means that fewer are selected as malnourished at the end than at the beginning and a plus sign conversely means a less favorable condition. It will be noted that the experimental group has a somewhat smaller percentage "selected" at the end on every basis except subcutaneous tissue score, while the controls have slightly poorer status on every basis except the weight score. Even for the subcutaneous tissue score, in which both groups had a poorer status at the end, the increase in the percentage selected is less for the experimental than the control boys, 9 and 22, respectively. Moreover on the one item for which the controls had improved, the weight score, their change for the better is less than for the experimental group. The findings of this portion of the study, then, so far as they go, are consistent with those for all preceding ones in showing a slight advantage for the boys who received the bananas.

The significance of the findings in relation to the adequacy of the diet in this institution and the light they throw on nutritional requirements will be discussed in the next article.

SUMMARY

The diet of 123 boys in an institution was supplemented with two to three bananas daily over a nine-month period and the effect on growth and other aspects of physical status observed in comparison with that of a control group in the same institution. A total of over 48,000 bananas was consumed during the period, or an average of 459 per boy. The bananas were well liked by the boys and were eaten with the same relish and dispatch at the end as at the beginning of the study.

On every basis of comparison the experimental boys had some advantage over the controls. This is shown by the following:

1. The experimental boys made a slightly greater mean gain in weight (0.90 pound) and height (0.15 inch) than the controls; a larger percentage of them equalled or exceeded the gains "expected" during the period (70 per cent vs. 44 per cent for weight, 71 per cent vs. 58 per cent for height); and their mean percentage gain during the year was significantly greater (127 for weight, 128 for height, vs. 105 for both weight and height).

2. In ossification of the bones of the wrist the experimental boys also showed slightly greater progress. This was true for mean gain in Carter index (3.2 vs. 2.9); percentage of boys making expected gains (49 vs. 36); percentage gain made by the group (79 vs. 74); and percentage of times gains of individuals exceeded those of the matched partner (51 vs. 41).

3. The Franzen measurements showed a similar trend. The boys who had received the supplement made greater mean gains in measurements of arm girth, subcutaneous tissue, and weight, and also in the scores for these items. The percentage of experimental boys "selected" as undernourished by these scores, moreover, decreased to a greater extent during the period than did that of the control group.

These differences do not in most cases attain statistical significance, but the fact that they were made on matched groups and that they are consistent in direction lend support to the belief that the differences are real ones attributable to the supplement.

The significance of the findings in relation to dietary requirements will be discussed in the next article.

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THE SUPPLEMENTARY VALUE OF THE BANANA IN INSTITUTION DIETS

II. Capillary Resistance and Reduced Ascorbic Acid in the Blood Plasma

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THIS report is a continuation of the preceding one,[1] in which the effect of a banana supplement on the growth and physical status of boys in an institution was described. The present one deals with the effect of the supplement on the vitamin C stores. Since the two or three bananas which the experimental boys received daily added from 340 to 510 international units of vitamin C (17-26 mg. of ascorbic acid or 34-51 Sherman units) to their diets over a fairly long period, it might be expected that their bodily stores of this vitamin would be increased over that of the control group, provided, of course, that the institution diet was previously suboptimal in this vitamin. Two measures were employed to determine whether this was the case: (1) the capillary resistance test and (2) the reduced ascorbic acid in the blood plasma of the boys in the two groups at the end of the study.

Capillary Resistance.—It is recognized that the capillary resistance test is affected by factors other than the body's store of vitamin C and that this test, therefore, cannot be used as a true measure of a given individual's state of saturation in respect to this vitamin. Nevertheless, as shown in a previous article,[2] there is evidence to indicate that it may be useful in large groups in determining relative saturation of the same individuals on different dietary regimes. The method was, therefore, employed in the present study for the light it might throw on the adequacy of the dietary in this institution for vitamin C. Four tests were made on the boys in both groups as indicated in Table I. The method used determined the negative pressure by the

Table I

Mean Capillary Resistance of Control and Experimental Groups on Four Tests (in Cm. Hg for Sum of Two Arms)

Test	57 pairs matched for resistance and age		All boys present for all four tests	
	Control	Exper.	Control 84 boys	Exper. 87 boys
I	32.3	32.3	33.1	29.9
II	26.6	29.6	26.7	27.7
III	31.1	32.7	30.7	31.9
IV	34.1	35.7	33.6	34.6

From the Department of Home Economics, University of Chicago.
The expenses of this study were defrayed in part by the United Fruit Company.

resistometer designed by Dalldorf[3] and the procedure was the same as has been fully described in a previous study.[2]

Inspection of the data obtained showed that individuals in both groups varied greatly in their initial resistance values: the extreme range was from 10 to 75 cm. of mercury for the sum of the two arms. Moreover, boys were occasionally absent at the times tests were made, and the records were not always complete. It was decided, therefore, that the fairest test of the results would be to use only subjects who were present for all four tests and to match these on the basis of original resistance values. There were found to be 71 partners who could be thus matched, and 57 of these were also well matched for age. Although these groups were considered the best to use for comparisons, calculations were also made for the total groups regardless of numbers, for all tests obtained at each period, and for several other classifications. Since the results were similar regardless of the basis of selection, they will be discussed only for the matched groups. The records for these boys were compared for (1) mean pressures on each of the four tests and (2) percentage of boys having high and low pressures on the successive tests.

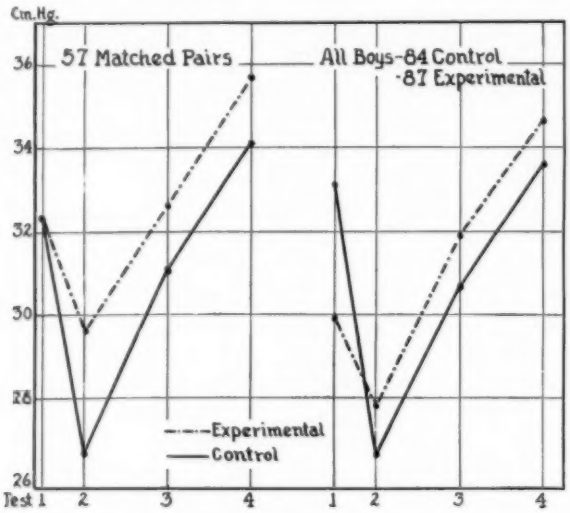

Chart 1.—Mean capillary resistance for control and experimental boys on each of four tests.

The mean pressures of the two groups on the several tests are shown in Table I and Chart 1. The two groups started with the same mean capillary resistance. Both groups went lower on the second test and rose again on the next two tests so that the values in June are well above those for October. Though the direction is the same, the level

for the experimental group is consistently above that of the control for all tests after the first, and conspicuously so for the second test. The percentage of boys having high and low values on the several tests reveals the same trend (Table II). This is best seen in Chart 2 by comparing the length of the bars on the extreme left for the two groups (40 cm. Hg or better), and likewise those on the extreme right (20 cm. Hg or less). It is noted that the percentage of boys having pressures of 40 cm. Hg or more for the two arms, equal for the two groups at the outset, has increased notably for the experimental group, from 33 to 53 per cent, while for the controls it has remained about the same, the values being 33 and 32 per cent. Conversely the percentage

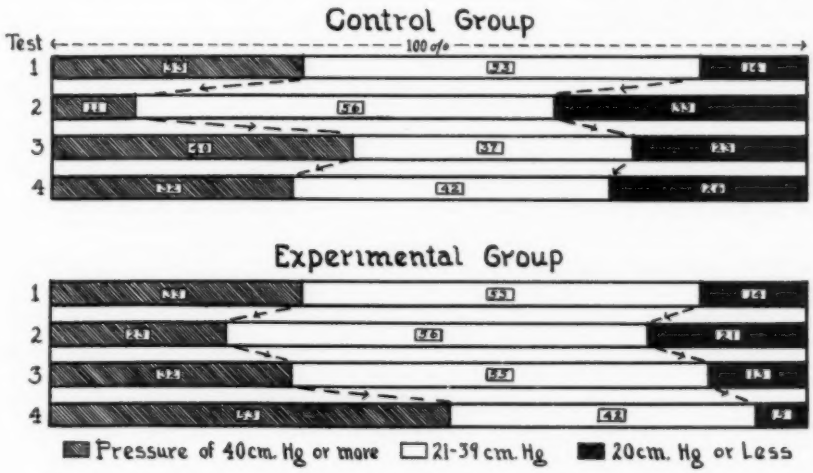

Chart. 2—Percentage of boys in control and experimental groups having high and low capillary resistance on each of four tests.

with low pressures of 20 cm. Hg or less has decreased for the experimental group from 14 to 5 per cent and has increased for the controls from 14 to 26 per cent. A fair proportion of individuals in the groups also followed the expected pattern, though the tendency was not so consistent as was observed in another institution where the anti-scorbutic vitamin was lower than in the one used for this study.[2] More than half of all individuals dropped lower on the second test (controls 60 per cent, experimental 56 per cent), and about the same proportion rose higher on the two succeeding ones. The drop for the experimental boys was, however, less, and their rise higher, with the result that 59 per cent of this group showed a gain from October to June, as against only 38 per cent of the controls whose readings had increased. There is no adequate explanation for the level in November being lower than in April, though it might be evident if a record of the entire year's dietary were available. The rise on the last test

coincides with the appearance of a few green things in the garden, and their inclusion to some extent in the diet. All the pressures were notably low, however, very few being better than 45 for the sum of both arms, and most of them much lower than this.

TABLE II

PERCENTAGE OF BOYS IN THE TWO MATCHED GROUPS HAVING CAPILLARY PRESSURES AT SPECIFIED LEVELS ON THE FOUR TESTS

PRESSURE CM. Hg SUM OF BOTH ARMS	CONTROL				EXPERIMENTAL			
	TEST 1	TEST 2	TEST 3	TEST 4	TEST 1	TEST 2	TEST 3	TEST 4
57 pairs matched for resistance and age								
40 and above	33	11	40	32	33	23	32	53
35	19	16	11	18	19	14	21	21
30	25	19	10	25	25	21	23	7
25	9	21	16	11	9	21	8	14
20 and less	14	33	23	26	14	21	16	5

To sum up, the differences in capillary resistance between groups were not large by any method of analysis, nor do they attain statistical significance. Nevertheless, the consistency of the direction of these differences together with the fact that they were obtained on matched groups suggests that though small the differences are probably real ones. It seems probable to the writers that the failure to observe more clear-cut differences may be due in part to the crudeness of the test, which registers pressures only in intervals of 5 cm. Hg. As pointed out in the preceding report, efforts should be directed toward refinement of the test itself, possibly along the lines of the instrument designed by Cutter,[4] before it can be used satisfactorily even for relative comparisons such as are here reported.

REDUCED ASCORBIC ACID IN BLOOD PLASMA

It has been shown by Abt, Farmer, and Epstein[5] and others that the amount of reduced ascorbic acid in the blood plasma can be used as an indication of the body's state of saturation with respect to this vitamin. They have also suggested levels that would probably indicate suboptimal states of saturation. Near the close of the year during which the present investigation was in progress, these workers reported the perfection of a micromethod by which determination of reduced ascorbic acid can be made on small amounts of blood.[6] It was decided, therefore, to make this determination on the boys in both groups at the end of the study even though the initial levels were unfortunately not known. If the dietary intake of vitamin C was suboptimal at the beginning, then it might be expected that the ascorbic acid* in the plasma of the boys who had been receiving the banana supplement would be significantly higher than that of the controls.

*Hereafter ascorbic acid will be used to mean reduced ascorbic acid.

The method of determination and details of procedure as outlined by Farmer and Abt[6] were followed precisely in order that the results might be comparable to those of their laboratory. To further this end, Farmer kindly sent one of his own laboratory workers skilled in the techniques of the method to assist in the determinations.* It was not possible in the short time available to make determinations for all boys. They were, however, made for a total of 182 boys, 93 in the group given bananas and 89 in the control group. Of these, 79 experimental and 65 control boys had been in their respective groups throughout the year and were included in the final analysis. The results of the determinations for these boys are reported on three bases: (1) mean values for ascorbic acid in the blood plasma of the two groups; (2) number and percentage of individuals having values at specified levels; and (3) relative values for partners matched for age and size. These have been determined both for the total number of individuals in each group for whom tests were made and also for 47 pairs, chosen from the matched group of 80 used for the comparisons of growth, for whom determinations for both of the partners had been made. This is, of course, a more or less arbitrary method of matching since no determinations of ascorbic acid were made at the beginning, and pairing could not be done on this basis. Without these initial blood levels, it seemed that the fairest comparison would be on the basis of age and size.

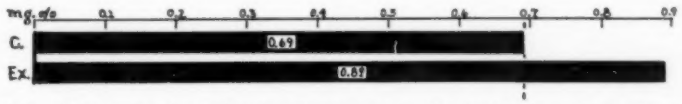

Chart 3.—Mean value for reduced ascorbic acid in the blood plasma of control and experimental boys.

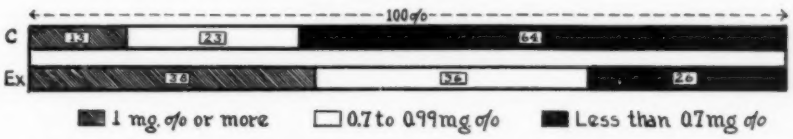

Chart. 4.—Percentage of boys in control and experimental groups having high, medium, and low values for reduced ascorbic acid in blood plasma.

Table III and Chart 3 give the mean ascorbic acid values for control and experimental groups, both for the matched and unmatched groups. The former is considered the more satisfactory basis and will, therefore, be considered chiefly in the discussion. The mean for the 47 experimental boys is 0.89 mg. ascorbic acid; for the controls 0.69 mg. This is a difference of 0.20 mg. ±0.034 in favor of the experimental group. This difference is, moreover, six times its probable error, and therefore, statistically significant.

*Miss Elizabeth Smith, to whom the authors wish herewith to express their appreciation.

TABLE III

REDUCED ASCORBIC ACID IN BLOOD PLASMA OF CONTROL AND EXPERIMENTAL BOYS AT END OF STUDY (EXPRESSED AS MILLIGRAMS PER 100 C.C. OF PLASMA)

	47 PAIRS FROM MATCHED GROUP			ALL BOYS*		
	CONTROL	EXPER.		CONTROL	EXPER.	
Total	47	47		65	79	
Mean	0.69	0.89		0.69	0.86	
Standard deviation	0.223	0.26		0.248	0.196	
Probable error	0.022	0.026		0.021	0.015	
Coefficient of variation	32	29		36	24	
Difference between means for groups			0.20			0.17
P.E. of difference			0.034			0.026
Ratio of difference to P.E.			5.9			6.5

*All boys present throughout the study who had ascorbic acid determined.

The percentage of individual boys having high values is likewise proportionately greater in the experimental group and those with very low values notably less than for the control boys. As seen in Table IV and Chart 4, 38 per cent of the experimental boys in the matched group have ascorbic acid values of 1.0 mg. per 100 c.c. of plasma, or better, as against 13 per cent of the controls having this high level. At the other end of the scale the picture is reversed. Using 0.7 mg. as the lower limits of the normal zone, as suggested by Farmer, 26 per cent of the experimental boys would be judged suboptimal, since their values were below this, while 64 per cent of the controls would be in the deficient group. A few boys in each group would even be classed in the scorbutic zone if values below 0.5 mg. as has been sug-

TABLE IV

PERCENTAGE OF CONTROL AND EXPERIMENTAL BOYS HAVING SPECIFIED LEVELS OF REDUCED ASCORBIC ACID IN THE PLASMA

MG. PER 100 C.C.	MATCHED PARTNERS*				ALL BOYS†			
	CONTROL		EXPER.		CONTROL		EXPER.	
	NO.	%	NO.	%	NO.	%	NO.	%
Total	47	100	47	100	65	100	79	100
1.0 and above	6	13	18	8	6	9	24	30
1.4 and over			2				3	
1.3-1.39								
1.2-1.29	4		4		4		5	
1.1-1.19			2				4	
1.0-1.09	2		10		2		12	
0.7-0.99	11	23	17	36	17	26	30	38
0.9-0.99	1		2		2		6	
0.8-0.89	5		7		7		13	
0.7-0.79	5		8		8		11	
Less than 0.7	30	64	12	26	42	65	25	32
0.6-0.69	12		7		21		18	
0.5-0.59	9		1		12		3	
0.4-0.49	9		4		9		4	

*Forty-seven pairs from 80 matched for height, weight, etc., for whom both partners had tests for ascorbic acid.

†All 96 experimental boys and 101 control boys who were present throughout the study remained in the same dietary group and received the tests.

gested by some workers be so considered; 9 of the control and 4 of the experimental boys fell below this level.

When the relative levels of blood ascorbic acid for the partners in the 47 pairs matched for age are examined, the superiority of the experimental boys is again evident (Table V). In 72 per cent of these pairs the experimental boy had the higher value, as compared with 21 per cent in which the control boy had the advantage. In the remaining 7 per cent the values were even.

TABLE V

COMPARISON OF LEVELS OF BLOOD ASCORBIC ACID FOR CONTROL AND EXPERIMENTAL PARTNERS IN 47 PAIRS MATCHED FOR AGE AND SIZE

	NUMBER	PER CENT
Total	47	100
Experimental partner has higher value	34	72
Control partner has higher value	10	21
Values even	3	7

It would appear then on all bases of anaiysis that the banana supplement helped materially in raising the level of reduced ascorbic acid in the blood plasma of the group who received it. It is significant to note, however, that the values were not high even for the experimental group, since only 39 per cent had readings of 1.0 mg. per 100 c.c. of plasma and above, and 24 per cent were even below 0.7 mg. There is no explanation as to why a few boys in the experimental group still had such low values. They may of course have eaten less of the potatoes and other vitamin C-containing foods in the regular diet so that the bananas may have been their chief source. If so, they might easily have had deficient intakes. The high values for some of the control boys are more easily explained. Larger intakes of potatoes, extra fruit which was occasionally brought in by parents, and possible access to rhubarb and a few other garden products which were just coming on would adequately account for them. In any case, the banana supplied during the winter months when other fresh fruits and vegetables were not available, provided a definite factor of safety in a diet otherwise questionable in its vitamin C content. It would seem evident, moreover, that the diet even with the supplement did not supply any excess of the antiscorbutic vitamin.

DISCUSSION

It is evident from the results of this study and the preceding one that the regular diet of the institution must have approached adequacy in most of the dietary constituents, for reasonably good growth and development occurred even without the supplement, and there was, moreover, no striking difference in the progress of the two groups. Nevertheless, it is also apparent that the diet was not quite optimal, for the group receiving the supplement showed some advantage over

the control group in every aspect of development which was measured. For growth in height, weight, arm girth, subcutaneous tissue, and ossification of the carpal zones, the differences were small and not always significant, but the consistency of their direction and the fact that they were obtained on matched groups lends support to the belief that the differences are probably real ones due to the supplement. The most pronounced results, however, were found in the measures of the vitamin C stores. For capillary resistance the experimental boys showed a definite rise during the year and a level higher than for the controls. The one outstanding difference in the entire study, however, was the significantly higher levels of reduced ascorbic acid in the plasma of the boys who received the bananas. This would indicate, as had been suspected, that the diet of the institution was suboptimal in vitamin C.

These findings must be interpreted in relation to the common dietary standards and to the diet of the institution both with and without the supplement. As indicated at the beginning, the various standards for food requirements of children which have been proposed differ markedly in many respects. This is explained by the fact that they are largely empirical, being based in the case of most of the constituents on inadequate experimental evidence. The diet of the institution was found to measure up to all minimum standards which have been proposed, but failed to equal the optimum standards for a number of constituents, especially vitamin C. The banana supplement, as shown in Table IV of Part I of this study, caused increases of 10 per cent or more in several of these constituents, and a 60 per cent increase in vitamin C. This brought the intake of most of the essentials up to the optimum standards or even above them. Some improvement in growth and physical status, moreover, resulted.

It is impossible to say which of the constituents of the supplement effected the improvement in growth. It may have been due to any one of the materials which it contributed in significant amounts—calories, vitamins A, B_1, C, G (riboflavin), and iron—or to the combined effect of several or all of them. The increase in the level of ascorbic acid in the blood was obviously due to the vitamin C in the supplement, and the slightly positive results in bone development might also be attributed to this factor.

In respect to the dietary standards, the results of this study would suggest that the amounts of the various dietary essentials which the diet of this institution provided when the supplement was added are not too high to allow for this age group (Table IV, Part I). These amounts are approximately the same, therefore, as those to be suggested later as tentative standards. It is possible of course, that some of these individual values may be higher than is actually essential, but since some improvement in physical status was effected when these

levels were attained, it would be safer to assume until further evidence is forthcoming, that most, if not all, of these constituents are needed in approximately these amounts. The chief value of this study, then, is the support it lends to the use of the higher standards for the various dietary essentials which have been proposed, rather than the minimum ones. It suggests in particular that the minimum standard of 600 international units for vitamin C should probably be abandoned and a higher allowance of at least 900 to 1,000 international units (50 mg. of ascorbic acid) employed. This is true, especially if all sources in the diet are to be counted, for there is always uncertainty as to the amount of the vitamin C lost in cooking, and staple foods as purchased in the market vary considerably in their content. In the present instance the values used in the computation of the amounts of the vitamin in the raw milk and in the cooked fruits and vegetables had to be arrived at somewhat empirically by the workers on the basis of their knowledge of the methods of cooking employed and the rations which the cows received. It is obvious that the values arrived at may therefore, be either too high or too low. The same uncertainties would, however, arise in any other situation in which computed values are used. For this reason the allowance in practical dietaries should be considerably higher than the amount shown experimentally to be sufficient when provided solely by one dependable source. A practical solution might be to include at least 600 units in a certain form, such as citrus fruit, tomato, or other known rich source, and to consider the amounts in the rest of the diet as supplements to this which will probably bring the total well up above this amount. The banana, although not a rich source of the vitamin, provides a fair and dependable amount. In institutions, such as the one studied, their liberal use would be a factor of safety in an economical and palatable form.

Reviewing the results of this study in connection with previous standards which have been proposed and with the small amount of experimental data available, especially those on calcium and phosphorus requirements, the writers are led to propose the following allowances as reasonable ones to use for boys from 10 to 12 years of age until more precise data on the needs for these years are obtained:

Calories	Protein	Ca	P	Fe	A	B_1	C	G (riboflavin)
	grams			mg.	S.U.	I.U.	I.U.	S.U.
2600-3200	75-85	1.0-1.5	1.2-1.5	14-18	4000-6000	300	1000	700

It will be noted that these allowances do not differ greatly from those of Steibeling nor from the higher levels suggested by other authorities save that 4,000 calories are allowed by some workers as the upper limit for energy needs. Even though some of these amounts may be higher than absolutely essential, they are safer to use until support for lower ones is forthcoming. They are, moreover, easily supplied

by the amounts of milk, vegetables, protein foods, fruits, and whole grain cereals, which are commonly advised as desirable to provide in the diets of growing children.

SUMMARY

This study, as reported in this and Part I, affords a small contribution to the problem of optimum versus adequate nutrition. It reports the results of supplementing the diet of approximately 100 boys living in an institution with two to three bananas daily over the period of a school year. The usual diet of the institution would be considered adequate in that it measured up to all minimum dietary standards which have been proposed. Nevertheless, it appeared to be susceptible of improvement, for the boys receiving the supplement showed consistently better growth and development by every measure of physical status which was applied than did the control boys who did not receive it. This was evidenced for growth in height, weight, arm girth, and subcutaneous tissue, for ossification of the carpal bones, and for the two measures of vitamin C stores used, namely, capillary resistance and reduced ascorbic acid in the blood plasma. The most significant differences were those for blood ascorbic acid. The experimental boys averaged 0.20 mg. per 100 c.c. of plasma higher than the control boys; this difference is six times its probable error and therefore significant.

These results indicate that the allowances for the various dietary essentials for boys of the ages here studied should approach the higher rather than the minimum standards which have heretofore been proposed, in particular for vitamin C.

Taking into consideration the findings of this study, previously proposed standards, and the small amount of experimental data available on food requirements, tentative allowances are proposed as reasonable ones to use for boys 10 to 12 years of age until more precise data on the needs for these years are obtained.

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RENAL DWARFISM DUE TO TUBULAR DYSFUNCTION

Clinical and Metabolic Studies of a Case

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CASES of rickets in which the etiological factor was recognized as vitamin D deficiency, but which did not respond to the usual therapeutic procedures, have been reported in the literature. The clinical severity alone does not distinguish these cases, as some severe cases of rickets with osteomalacia and osteopsathyrotic changes respond to vitamin D therapy. In addition to the resistance to therapy, these cases have a different clinical course, which suggests some basic disturbance in addition to the avitaminosis. For example, rickets is recognized as a complicating syndrome in cases of nephritis (Mitchell[8]) and in liver disease in children (Gerstenberger).

Recently there have been reported cases from France and Switzerland with apparently marked rachitic changes, which did not respond to vitamin therapy and which were not identical with renal rickets. Debré[2] and de Toni,[13] who first observed such cases, differentiated them from renal rickets. Fanconi[4] was the first to analyze the condition on the basis of clinical and metabolic investigations as one of distinct kidney dysfunction.

We had occasion to observe a similar case recently.

Family History.—Parents were unrelated; father, 28 years old, was healthy, and mother 29 years old, healthy. There was no kidney disease in the family.

Personal History.—The patient, the first child, was born after normal labor, and weighed 3,850 grams at birth, weaned at 8 months, and fed subsequently with milk mixture. The first tooth appeared at 6 months. The child was able to stand at 8 months and began to walk at 10 months. At 18 months the child stopped walking, became obviously weaker, and finally could not stand. The family physician prescribed cod-liver oil. Within a period of five months, the child was given 1¾ liters of cod-liver oil, 1 bottle of vigantol, 1 bottle of infadin-bioklein, and calcium, and was treated many times with ultraviolet rays. Since despite treatment the child did not improve, the growth remained retarded, and clavicular deformities appeared, the family physician advised hospitalization.

Examination on admission revealed a 2-year-old delicate child with light soft smooth hair, blue eyes, and thin perspiring skin, through which the subcutaneous veins shimmered. The child weighed 8,700 gm., and was 75 cm. long, approximating the values for a 1-year-old child (Fig. 1). Striking thoracic deformities were present. The thorax was retracted laterally with wedge-shaped prominence of the sternum and costosternal junction. The inferior margin of the thorax was prominent. Both collar bones buckled symmetrically anteriorly (Fig. 2). The extremities were curved and the distal endings of the epiphyses were swollen. Marked hypotonus of the

From the Second German University Children's Clinic in Prague, under the direction of Dr. Berthold Epstein.

muscles of the extremities was present. The tendon reflexes were normal. The child cried out, obviously with pain, on any attempt to lift him into one's arms. The roentgenograms revealed curving of the bone with severe decalcification. This deficiency in x-ray opaque substances was so marked in many areas, that the contours of many epiphyseal endings and ossification centers could not be identified (Fig. 3). The substantia spongiosa appeared glassy, whereas the substantia compacta was recognizable as thin stripes. There were fractures of the radius, ulna, and fibula, as a result of marked fragility of the bones (Fig. 4). (We were able to observe by repeated x-ray examination the slight tendency of the fractures to heal.) The more typical rachitic changes were not demonstrable. In the hand there were two ossification centers in the capitate and hamate bones corresponding to those of a 1-year-old child. Both ossification centers of the foot were decalcified almost beyond recognition.

The heart and lungs were negative except for a tachycardia of 130 per minute and increased respiratory rate of 40 per minute.

With regard to the clinical course, it should be mentioned in advance that the nourishment of the child in the beginning was difficult because of the absolute anorexia. The child took only fluid nourishment. At times constipation was marked. Now and then there were unexplainable transient fever episodes up to 38° C.

Fig. 1.

Fig. 2.

There was a persistent presence of sugar in the urine. In an almost constant daily urinary output of 100 c.c., there was usually 2 per cent sugar, which was identified as glucose by fermentation test. The specific gravity was constantly high, about 1.030. Traces of acetone were present only twice, while diacetic acid was never found. Occasionally, traces of albumin were demonstrable with sulfosalicylic acid. Millon's test for thyrosin was occasionally positive. The urinary sediment, except for a few granular casts, was not unusual. The cloudiness of the constantly alkaline urine disappeared with the addition of large amounts of acetic acid.

Although the child had received small amounts of fruit, we wished to exclude a vitamin C deficiency because of the generalized tenderness. This was suggested by Czerny's[1] description of an unusual form of vitamin C deficiency during the World War, which was characterized by osteoporosis and pathologic fractures. These

cases, according to Czerny, responded therapeutically to carrot juice. Therefore, we made a vitamin C tolerance test according to the method of Harris and Ray. After the injection of 100 mg. of vitamin C in the form of redoxon (Hoffmann-LaRoche), 28 mg. was recovered in the urine during the next three hours (the vitamin C in the urine was determined according to the method of Martini and Bonsignore). This excretion corresponds to a normal tissue saturation with vitamin C.

Although vitamin D insufficiency was most improbable, considering the history of 5 months' treatment for rickets, we once more attempted vitamin D treatment. In spite of a concentrated vitamin D therapy (vigantol—Stoss), there was neither clinical nor roentgenologic improvement.

There was 1.4 mg. per cent inorganic phosphorus in the serum, which was the lowest serum phosphorus concentration we ever encountered in the clinic in a child. The serum calcium on the other hand, was markedly elevated and equal to 14 mg. per cent. The fasting blood sugar varied repeatedly between 50 and 65 mg. per cent.

Fig. 3.

Fig. 4.

There were 5.5 million red cells with 70 per cent hemoglobin. The white cell count was 15,400; of these 57 per cent were neutrophilic leucocytes, 2 per cent eosinophilic leucocytes, 1 per cent stab cells, 39 per cent lymphocytes, and 1 per cent monocytes. Sedimentation rate according to the micromethod of Linsenmaier was 33 mm. in one hour, or approximately twice the normal value.

This clinical condition probably corresponds to the early infantile nephrotic dwarfism with glycosuria and hypophosphatemic rickets, which Fanconi[4] described on the basis of three cases, and to the cases described by de Toni,[13] Debré,[2] Ullrich,[14] Schlesinger,[12] Guild,[15] and Pineus.[16] This condition is probably a distinct clinical entity and is to be differentiated from the usual renal rickets. Following Fanconi's clinical and metabolic studies of the condition,[3, 4] similar cases were discovered

by Fornara[7] and Nobécourt;[9] thus we may conclude that in the future this clinical condition will be observed more frequently.

Our case from the clinical standpoint is somewhat unusual. The clinical course at first was similar to the cases described by other observers, that is, it developed in an apparently healthy child who rather suddenly became unable to stand or walk. We were led to investigate the skeletal system because of the pain on lifting the child. As in the other cases described, there were anorexia, constipation, and thermolability. The urine output, however, differed strikingly from the cases described by Fanconi in that albumin was present only in traces and there were very few casts. In Fanconi's cases (e.g., Case 3) the urinary sediment occasionally contained epithelial cells, leucocytes, fat cells, red blood cells, and sometimes numerous granular casts, and there was up to 3 mg. per cent albumin present. We could not regard our case as typically nephrotic on the basis of the urinary findings. In addition, the specific gravity of the urine was high in a very small output of urine, a condition not found in the other cases. Nevertheless, we intend to show that in our case there was also an underlying disturbance in kidney function.

Although the diminished urinary output was more characteristic of nephritis than of nephrosis, the former possibility was soon eliminated by the dilution and concentration test wherein the range of specific gravity was 1.003 to 1.030. After the administration of 250 gm. of tea sweetened with saccharin, only 40 c.c. of urine was excreted after two hours, and only 80 c.c. after four hours. In spite of this small urinary output, the urine was diluted to 1.003 after two and one-half hours. In the subsequent concentration test the urine reached a specific gravity of 1.030 after four hours of thirsting. The blood nonprotein nitrogen was 32 mg. per cent, and the blood pressure 80 mm. The eyegrounds were normal. When these normal findings are considered, the case could not be regarded as an inflammatory kidney disturbance, regardless of the markedly reduced urinary output. Neither could the case be looked upon as a simple nephrosis, because the albuminuria was slight, no double refractile bodies were present in the urinary sediment, the urinary sodium chloride quotient was normal, and there was no edema. On the other hand, the metabolic studies which we made convinced us of the presence of a kidney tubule dysfunction. The elevated specific gravity of the urine was probably due essentially to the glycosuria and characteristic salt excretion.

The first metabolic studies were made shortly after admission of the child into the hospital, when he was receiving a normal mixed diet of milk, vegetables, fruit, and meat. (A more complete analysis of the diet is found in Table I.)

From a clinical standpoint the rapid deep respiration, especially during sleep, the very striking thirst, and the reaction of the child

TABLE I

METABOLIC STUDIES

LENGTH OF TIME OF EXPERIMENT	METHODS	DIET 5 DAYS	FECES 3 DAYS	URINE 3 DAYS
Amount per day		1.010 gm.	40 gm.	115 c.c.
Weight of desiccated feces			7.15	
Protein	Folin	2.73	0.32	3.67
Carbohydrate	Hagedorn-Jensen	107.6		2.64 gm.
Fat		32.75		
P_2O_5	Bell and Doisy	1.51	1.6	0.18
P_2O_5 in per cent of diet			109.91	12.5
Calcium	Tisdall and	0.917	0.75	0.018
Calcium in per cent of diet	Kramer		81.52	1.95
Sodium	Barrenscheen	0.439	0.054	0.248
Sodium in per cent of diet	Folling (Harn)		12.36	56.62
Chloride	Vollhard	0.98	0.128	0.306
Chloride in per cent of diet			13.06	31.22
Potassium	Tisdall and	2.337	0.343	0.389
Potassium in per cent of diet	Kramer		14.65	16.61
Total basic valencies		17.9		
Total acid valencies		7.4		

to any infection, suggested acidosis.* During an episode of tonsillitis, the child, who was usually alert, became apathetic with distinct Kussmaul respiration. This could be substantiated by determining the excretion of organic acids according to the method of Palmer and Van Slyke. The child excreted from 29.7 to 42.4 c.c. 1/N organic hydrochloric acids per kilogram of body weight, whereas, according to Hottinger[7] and Fanconi,[4] approximately 12.5 c.c. per kilogram is excreted by the normal child.

The morning hypoglycemia of 50 to 65 mg. per cent and the morning sweats were probably associated with the unstable acid-base metabolism.

TABLE II

TABLE OF BALANCES FROM THE FIRST EXPERIMENT

	MG. PER CENT IN SERUM	GM. IN DIET	GM. IN FECES	GM. IN URINE	BALANCE	
					ABSOLUTE	PER CENT
Nitrogen	32.0 N.P.N.	2.73	0.32	3.67	-1.26	-40.7
Carbohydrate	182.0 Blood sugar	107.65		2.64		
P_2O_5	3.36	1.51	1.6	0.18	-0.27	-17.8
Calcium	14.1	0.917	0.75	0.018	+0.14	+16.9
Sodium	237.6	0.439	0.054	0.248	+0.136	+31.2
Chloride		0.98	0.128	0.306	+0.046	+ 4.6
Potassium	8.87	2.337	0.343	0.389	+1.6	+ 6.8

It is evident from Table I that the child's diet contained 17.9 basic valencies to 7.4 acid valencies. In Table II the mineral metabolism is summarized. There was a negative balance for phosphorus, a weakly

*A monograph on rickets by Eliot and Park (Hagerstown, Md., 1938, W. F. Prior Company) has just appeared, wherein the authors describe a case of the de Toni-Fanconi disease complex with 30 volumes per cent carbon dioxide combining power. (The same by Guild.[15]) The acidosis could not be corrected with either sodium bicarbonate or sodium lactate.

positive balance for chloride and potassium, and a strongly positive balance for sodium and calcium. Most striking is the negative phosphorus balance of 122 per cent excretion as compared with the phosphorus intake. This could explain the acidosis as due to loss of the phosphate blood buffers, and the demineralization of the bones. This combination of intermediary acidosis and decalcification is usual in rickets. In our case the Howland-Kramer product of calcium and phosphorus in the blood was 19.88, which is typical for active rickets, in which the product is usually less than 30. The György calcium phosphorus quotient, which is normally 2 and in rickets is elevated to 3.5, reached the unusual value of 10.4 in our case. The maximum phosphorus excretion was with the feces, as is characteristic of rickets.

In spite of the fact that the described metabolic changes are so similar to those of rickets, the typical roentgenologic changes of rickets, the osteoid zones and swelling of the epiphyseal endings, were absent. It must be recalled, however, that Pearson distinguished three different types of rickets. The atrophic type, the most common, consists of typical rachitic changes only at the line of ossification, whereas the pathologic fractures, thin cortex, and osteoporosis recall malnutritional osteopathia; the second type consists of active rachitic changes; and the third type which Pearson describes as the "spotty honeycomb" type is characteristic of active late rickets. The osseous changes in our case may easily be classified under the first type.

Our doubt as to assuming the rickets to be alone responsible for such bone changes is justified because of the child's failure to respond to the usual therapy, while the combination of acidosis and decalcification is not pathognomonic of rickets.

In 1917 Schloss[11] regarded rickets, not as an isolated disturbance of phosphorus and calcium metabolism, but as a general form of disturbance of growth in infants to be attributed to disturbances in the internal nutritional processes of growing cells. Today we recognize acidosis and decalcification in a variety of clinical conditions, as celiac disease, osteomalacia, and malnutritional osteopathia.

We felt that in our case, in order to treat the metabolic disturbance, it was necessary to give the child a diet consisting essentially of alkaline balances. In planning such a diet, we were guided more by the biologic effect of the food rather than its actual hydrogen ion concentration. It is very difficult, however, to devise such a diet, as the chemical reaction of the tissues does not necessarily parallel that of the blood. In addition, in the organism the bases always combine with distinct acid radicals. It is essential, therefore, in formulating such a diet, to regard separately the various ions it contains. For example: sodium or potassium may be made predominant in the diet. These bases are biologically antagonistic and can displace each other.

We gave the child an alkaline diet in which potassium predominated, believing with Schloss that the disturbance was a primary dysfunction of the cell which sodium would only aggravate. Therefore, our patient's diet consisted essentially of vegetables and fruits. The child received no meat, little fat, and never purely carbohydrate foods. A typical daily menu consisted of cocoa and banana in the morning, vegetable purée with potatoes for lunch, cocoa in the afternoon, and banana purée in the evening. We preferred banana to all other fruits because it contains almost ten times as much potassium as sodium. For similar reasons we preferred potatoes to other vegetables. As is shown in Table III, the second diet contained 3,346 mg. of potassium as compared

TABLE III

METABOLIC STUDIES

TIME OF EXPERIMENT	METHOD	DIET 3 DAYS	FECES 3 DAYS	URINE 3 DAYS
Weight per day		1.041 gm.	40 gm.	88 c.c.
Weight of desiccated feces			7.9	
Protein	Folin	2.576	0.43	2.6
Carbohydrate	Hagedorn-Jensen	184.53		2.4 gm.
Fat		43.46		
P_2O_5	Bell and Doisy	1.79	0.007	0.155
P_2O_5 in per cent of diet			0.39	8.65
Calcium	Tisdall and	0.94	0.36	0.014
Calcium in per cent of diet	Kramer		38.29	1.48
Sodium	Barrenscheen	0.752	0.015	0.31
Sodium in per cent of diet	Folling (Harn)		1.98	41.22
Chloride	Vollhard	1.108	0.095	0.564
Chloride in per cent of diet			8.56	50.9
Potassium	Tisdall and	3.346	0.131	0.304
Potassium in per cent of diet	Kramer		3.91	9.08
Total basic valencies		50.57		
Total acid valencies		3.52		

TABLE IV

BALANCES FROM THE SECOND EXPERIMENT

	MG. PER CENT IN SERUM	GM. IN DIET	GM. IN FECES	GM. IN URINE	BALANCE	
					ABSOLUTE	PER CENT
Nitrogen		2.57	0.49	2.6	-0.52	-20.2
Carbohydrate		184.53		2.4		
P_2O_5	4.8	1.79	0.007	0.155	+1.62	+90.8
Calcium	17.59	0.94	0.36	0.014	+0.56	+60.2
Sodium		0.752	0.015	0.31	+0.43	+56.7
Chloride	361.62	1.108	0.095	0.564	+0.4	+40.5
Potassium	20.83	3.346	0.131	0.304	+291	+87.15

with 2,337 mg. in the diet on admission. There was no marked difference in protein, carbohydrate, fats, and other minerals. By determining the mineral balance with this diet, striking changes were made evident. The 17.8 per cent negative phosphorus balance became positive, reaching even 90.8 per cent; the calcium balance increased from 16.9 to 60.2 per cent; the sodium from 31.2 to 65.7 per cent; the

chloride from 4.6 to 40.5 per cent; and the potassium from 6.08 to 87.15 per cent. All of the minerals were therefore strikingly retained. From the above figures it is obvious that phosphorus and potassium were retained to a greater degree than calcium, sodium, and chloride. This is the usual antagonistic grouping of minerals encountered in many studies. Another striking change resulting from this diet is shown in Tables V and VI. Most of the phosphorus is excreted in the urine rather than in the feces, a change which occurs typically in healing rickets.

TABLE V

PERCENTAGE OF MINERAL EXCRETION DURING THE FIRST EXPERIMENT

	FECES	URINE
P_2O_5	89.9	10.1
Calcium	97.65	2.56
Sodium	17.9	81.84
Chloride	29.49	70.5
Potassium	46.98	53.28

TABLE VI

PERCENTAGE OF MINERAL EXCRETION DURING THE SECOND EXPERIMENT

	FECES	URINE
P_2O_5	4.32	95.67
Calcium	96.25	3.76
Sodium	4.61	95.38
Chloride	14.41	85.58
Potassium	30.11	69.88

The effect of this high potassium diet on the acidosis was shown in a change in the amount of organic acids excreted. Only 24 c.c. 1/N hydrochloric acid was excreted as compared with 42.4 c.c. The hydrogen ion concentration of the urine changed from 7.2 to 6.8; the blood serum phosphorus increased from 1.4 to 2.1 mg. per cent; and the calcium from 14.1 to 17.59 mg. per cent. In addition, after six weeks of this diet, the glycosuria disappeared, and the blood sugar increased to 85 mg. per cent. We did not make glucose or adrenalin tolerance tests because in two cases similar to ours death was reported to have resulted from such tests. We did not risk the insulin tolerance test because of the hypoglycemia.

Of special importance in our case is the calcium. Both the high blood serum calcium and decalcification might suggest a disturbance similar to Recklinghausen's ostitis fibrosa. In our case the serum calcium was 14.1 mg. per cent on the first diet, and it increased to 17.59 mg. per cent on the second diet. With both diets there was a positive calcium balance, the calcium being excreted with the feces. This contrasts, however, with the negative calcium balance and calcium excretion in the urine in ostitis fibrosa. In osteomalacia there is also increased serum calcium with a negative calcium balance. In many such

cases, and in cases of rickets, hypertrophy of the parathyroids has been found. This hypertrophy has been regarded as a secondary compensatory phenomenon. In our case hyperfunction of the parathyroids is very unlikely because of the positive calcium balances and normal excretion of calcium in the feces. It is interesting to note that the elimination of calcium occurred with the feces despite the acidosis. We have, therefore, an abnormal distribution of calcium between the blood and tissues which does not fit into any known category of calcium metabolism disturbance.

We should like to explain the kidney disturbance in our case with the help of the modern knowledge of the physiology of the kidney, several of our own experiments, and the literature about similar cases. Fanconi is of the opinion that, in cases such as the one we are describing, only the function of the kidney tubules is altered, with disturbed reabsorption of water and salts. He was able to substantiate this interpretation as a result of an autopsy of such a case, in which the tubular epithelium exhibited vacuolar degenerative changes. Fanconi's studies of the mineral balances in his own cases suggested faulty tubular reabsorption of such substances as sugar, phosphorus, and potassium. This excretory disturbance appears to be characteristic of the clinical syndrome under discussion. Nevertheless the question remains whether the increased urinary excretion of biologically electronegative substances like sugar and these salts is due to the same lesion.

According to Ludwig's and Cushny's theory of urine formation, the glomerular urine has the same crystaloid concentration as the blood plasma. The definite urine is formed in the tubules as a result of reabsorption of water and threshold substances. It has become possible to study the function of distinct portions of the tubules. As a result of Popper's investigations,[10] it has become possible to demarcate the extent of glomerular filtration and tubular reabsorption. Thus the fraction of urine creatinine over plasma creatinine multiplied by the urinary output per unit of time equals the glomerular filtration, because creatinine is a nonthreshold substance. The extent of tubular reabsorption equals the glomerular filtration minus the urinary output. The average glomerular filtration by an adult is 80 to 180 c.c. per minute, of which 98.3 to 99.8 per cent is normally reabsorbed in the tubules of the fasting individual. Popper, Mandl, and Mayer were able, with the aid of a modified and more exact method of plasma creatinine determination, to determine the minimum variations of the plasma creatinine. This makes it possible to distinguish glomerular and tubular insufficiencies. In our case the urine creatinine was 50 mg. per cent, and the plasma creatinine, 1.43 mg. per cent. Since the urinary output per minute was 0.3 c.c., the glomerular filtration equalled 10.7 c.c. Of this, 97.2 per cent was reabsorbed in the tubules. This corresponds to Popper and

Mandl's findings[10] in cases of acute nephritis and beginning nephrosis, which they regard as a serous capillary inflammation.

In contrast to this disturbance, in cases of contracted kidneys, there is an essential reduction in the percentage of reabsorption, which would indicate a disturbance in tubular function. In our case we may conclude that the reabsorption of biologic electropositive water by the tubules was undisturbed. This corresponds closely with the positive balance with the other biologic electropositive substances, especially of sodium, in Fanconi's studies, as well as in ours. We assume, by exclusion, a disturbance in the tubular reabsorption only of biologically electronegative substances like sugar, phosphorus, and potassium. Popper and Mandl[10] found similar normal reabsorption of water following the administration of phlorizin in adults with normal kidneys. Further evidence of the similarity of our case with that of phlorizin-injured kidneys is the fact that the quotient of urine and blood sugar in our case corresponded with that in individuals after phlorizin administration. We may conclude, therefore, that phlorizin diabetes and the glycosuria in our case have the same basis.

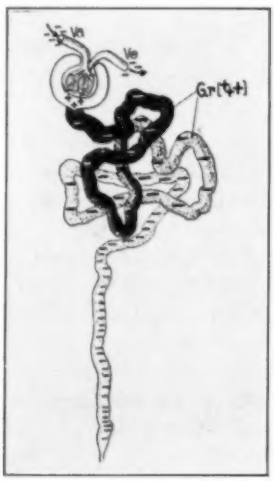

Fig. 5.

According to modern physiology, the protoplasm of the tubular epithelium is charged negatively, but contains in the proximal convoluted tubes electropositive granules which predominate over the negatively charged protoplasm (Keller[17]). As a result, negatively charged substances like urea, sugar, potassium, and phosphorus are reabsorbed in this region. The positively charged substances like water, sodium, and chloride are reabsorbed in the lower portion of the tubules, which has a predominantly negative charge (Fig. 5). The effect of phlorizin is ex-

plained in that it has a stronger negative charge than sugar and that it binds and neutralizes the electropositive granules of the tubuli contorti, with the result that the reabsorption of sugar is hindered. It would appear that the infantile nephrotic glycosuric dwarf with hypophosphatemic rickets (Fanconi[4]) is the clinical analogue of experimental phlorizin poisoning.

There remains only to explain the therapeutic effect of a higher potassium diet in this clinical condition. The same question pertains to the beneficial effect of the banana diet in cases of celiac disease with which Fanconi has been able to eliminate the intermediary acidosis and demineralization. In these functional disturbances the potassium seems to activate the protoplasm of the cell. György[6] determined that potassium, in contrast to sodium, stimulates the cellular respiration.

We are not in a position to predict whether the beneficial effect of the higher potassium diet in our case on the metabolism will last or whether the skeletal changes will improve. Since, however, the prognosis in such cases has been invariably fatal regardless of any previous form of therapy, we consider the dietetic effect obtained in our case of significance.

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CEREBRAL VASCULAR LESIONS IN NEWBORN INFANTS AND YOUNG CHILDREN

With the Report of Forty Among One Thousand Necropsied Cases With Spontaneous Vascular Encephalopathy

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THE relative frequency of spontaneous cerebral vascular lesions in the young is not generally realized. Cerebral lesions in the newborn infant are too universally attributed to the trauma of childbirth with insufficient consideration of other contributory causes. The pathology and treatment of these early cerebral injuries have received considerable attention with resultant greater realization of their frequency and application of earlier helpful aid. Pathologic studies in newborn infants showing cerebral injury indicate the use of forceps and other manipulative aids to delivery is not as provocative of trauma as spontaneous precipitate or short, rapid labors. This fact is particularly evidenced by the frequent finding of hemorrhage from the rupture of cerebral vessels accompanied by tearing of the tentorium cerebelli. The dura mater encephali lines the cavity of the skull like a periosteum and in children is everywhere firmly atttached to the bone.[1] The sudden anterior-posterior pressure from rapid passage of the head through the birth canal forcing a correspondingly sudden increase in the lateral diameters of the skull is the mechanism most potentially provocative of tearing and rupture of these membranes with their neighboring and contained vessels and sinuses. Studdiford[2] discussed the pathology of fatal cerebral birth injuries describing the most common mechanisms. Anterior-posterior compression with tension on the great cerebral vein and its branches may result in small subdural hemorrhages. Excessive abrupt lateral compression causes large tears in the substance of the falx with cerebral injury occurring from loss of cranial support. Hemorrhage from the small subarachnoid veins may follow edema of the scalp, and thin-walled veins beneath the floor of the lateral ventricles may rupture with ensuing subventricular or intraventricular hemorrhage.

The frequency of intracranial birth injuries is indicated in Nattrass'[3] report of 46 per cent in 107 consecutive necropsies. Galloway[4] reports fatal cerebral hemorrhage in 30 per cent of 3,011 autopsies from European clinics, in 28 per cent of 617 in American hospitals and in 34 per cent of 291 autopsies in his study. He states 40.6 per cent of full-term babies dying at birth show cerebral hemorrhage. Extradural hemorrhages resulting from birth trauma are discussed by Lefkowitz[5] and Dietrich[7] offers an enlightening exposition of the pathology and incidence, with illustrative cases, of hemorrhage in the newborn infant.

In 1924, Sharpe and Maclaire[8] revealed some rather important facts about intracranial hemorrhage in the newborn infant. They found that death from intracranial hemorrhage in the newborn infant results from extensive hemorrhages and cerebral edema unless the extravasation of blood can be entirely absorbed by the natural means of excretion without any resultant organizing residue. The unrecognized and therefore improperly treated cases of milder intracranial hemorrhage develop later, in a large per cent, some form of cerebral spastic paralysis with or without mental impairment.

They showed that cerebral spastic paralysis due to intracranial hemorrhage should be different from Little's disease, the latter including only those cases due to cerebral agenesis and meningoencephalitis.

Pediatricians, neurologists and neurosurgeons usually see the cases late as chronic conditions, when spastic paralysis in its various forms has already developed and when conditions at least can only be improved, whereas, it is the obstetrician who sees these cases in the acute stage. The acute cases with mild signs or no signs at all are overlooked.

In a study to detect the apparently signless intracranial hemorrhages in the newborn infant they found that 13 out of 100 consecutive deliveries were afflicted with intracranial hemorrhage of varying degree. Low forceps and syphilis were not found to be the etiological factors causing the intracranial hemorrhages. Likewise the coagulability of blood did not appear to be an important causative factor. Spinal puncture as a diagnostic and therapeutic measure was proved in this series to be a safe procedure in the absence of pre-existing shock. It was discovered that apparent signs indicative of an acute intracranial hemorrhage and cerebral edema can be confirmed or disproved by early spinal puncture.

Spinal puncture was advocated as a safe routine procedure in suspected cases having the mildest signs of intracranial hemorrhage and cerebral edema, provided the operation is done within the first seventy-two hours, and is a valuable aid to the natural means of their absorption.

Repeated spinal drainage was advocated in cases of bloody spinal fluid. The intervals between drainage were from six to twenty hours, depending on the varying degrees of pressure. They found that if repeated spinal puncture failed to diminish progressively the blood concentration and the pressure of the spinal fluid, then a modified subtemporal decompression and cranial drainage were indicated.

The fact that previous figures state that about 9 out of every 100 deliveries suffered intracranial hemorrhage and that they discovered intracranial hemorrhage present in 13 per cent of their cases indicates that the intracranial lesion is more common at time of birth than has previously been conceived. They do not believe it sufficient to employ remedies intended to increase the coagulability of the blood without measures being undertaken for the purpose of removing from the cerebrospinal system the blood which has already escaped from the ruptured vessels.

In 1925, Sharpe and Maclaire[9] reported a case of intracranial hemorrhage in a newborn infant as a cause of hydrocephalus. Bloody spinal fluid was found by spinal puncture soon after birth. Eight weeks later distinct signs of hydrocephalus developed. He suggests that the clot forming from the intracranial hemorrhage organized and occluded the normal channels of excretion for the cerebrospinal fluid. The back pressure increased until it started to distend the ventricles and the bulging brain secondarily forced the individual cranial bones to separate, thus resulting in a typical picture of hydrocephalus.

Failure of normal development of intracranial structures, as demonstrated in prematurity and agenesis, offers greater response to less trauma despite the possible smaller size of the child's head. Patten[10] has maintained that many of the cerebral birth conditions are primarily due to failure of development of the brain, particularly in the realm of myelinization. Further evidence of this contention was elaborated in a study of thirty infant brains, both premature and at term, by Patten and Alpers.[11] There were found punctate and large hemorrhages in the subependymal region involving the matrix area causing destruction of and interference with spongioblasts which would eventually be concerned in the deposit of myelin by virtue of their development into oligodendroglia. The factor of subarachnoid hemorrhage as a cause of these cerebral conditions was discounted as compared with the more vital hemorrhage in the subependymal region, the cause of which is not clear.

Hypoplastic vessels, as found in patients with the so-called "thymic syndrome,"[12] are more readily subject to rupture, even spontaneously, than normal arteries.

Spontaneous cerebral hemorrhage has been found frequently in youths and children without apparent cause, but careful investigation in some instances has disclosed the small heart and vascular hypoplasia in the major and cerebral vessels of the so-called thymic syndrome. Some autopsy surgeons[16] believe they are able to recognize in the artistic facial features and body mold of these patients attributes of this condition and frequently correctly predict the cerebral hemorrhage later found at necropsy. From the analysis of the data collected by the status lymphaticus investigation committee in 1931,[14] there was rather general agreement that status lymphaticus does not exist, but this does not preclude that a hypoplastic cardiovascular condition may exist which predisposes to cerebral hemorrhage in the young.

Kolisko[17] reported a ventricular hemorrhage in a man 20 years old with a "lymphatic constitution." Narrowing of the arterial system had led to secondary cardiac hypertrophy and beginning endocarditis.

Pilez[19] reported a 30-year-old woman, previously in fair health except for repeated sore throat and anemia, who one evening suddenly became unconscious and vomited. Consciousness was soon regained when the

patient complained of severe pain in the eyes and was partially paralyzed in the left arm and face. She lapsed into unconsciousness and had a tonic spasm, first in the left arm and then in the right extremities. Death occurred in a few hours. At necropsy a large hemorrhage was found in the right frontal lobe which had ruptured into the ventricle. No aneurysm was found in spite of minute scrutiny. The arterial system was extraordinarily narrow with thin and elastic vessel walls. The ascending aorta barely admitted the index finger. The carotids and femorals were no larger than the radials in a normal person. The heart was very small. The intima of all vessels was smooth. There was no enlargement of the thymus or lymphatic glands. Histologic examination revealed fatty degeneration of the heart muscle and moderate sclerosis of the small vessels in the kidney. There was no evidence of arterial disease in the brain and no miliary aneurysm; the congenitally overdelicate vessel had simply ruptured.

Kowitz[20] previously had reported a series of cases with ventricular hemorrhage mainly in youthful individuals.

Waldbott and Anthony[21] reported a follow up study of 24 of 30 children whose condition had been diagnosed by the roentgen method as "enlarged thymus." They found these 24 children were afflicted with allergic disease as evidenced by the histories and skin tests. The possible relationship of thymic enlargement to anaphylactic death was discussed and thorough allergic studies were suggested in cases with x-ray evidence of enlarged thymus.

Waldbott[22] selected 34 cases where no adequate cause of death had been determined among 102 patients in whom status lymphaticus had been diagnosed by various pathologists. In 11 of the 34, death had occurred without previous illness nor any pathologic lesion to indicate the presence of previous illness. In 16, minor, usually nonfatal incidents preceded death. In 7, a syndrome developed manifesting dyspnea, stridor, fever, and shock. In 17, petechial hemorrhages involved the heart, pleura, lymph glands and various other viscera. The brain was involved by petechial hemorrhage in but one, but there was edema of the brain noted in 12 of the 34. Comparison of the pathologic process with that reported in anaphylactic shock in man reveals a close resemblance, if not a complete identity, in the two conditions. On the basis of these findings it is believed death may be the result of primary anaphylactic edema of the lungs and ensuing asphyxia. Such a theory can be upheld only if one assumes that anaphylactic shock may occur from incorporation into the body of nonprotein substances and that absorption of shock-producing antigen may take place by ways other than by injections.

Intrauterine fetal infections, about which little is known, are surely contributory factors in some instances of cerebral vascular pathology, as displayed in the cerebral hemorrhages found in congenital syphilities where endarteritis has made the vessels more liable to rupture.

From the first hour of birth the child is subject to infections and in rare instances encephalitic processes may follow variola, vaccinia, varicella, and possibly mumps and measles. The assumption, however, that these encephalitic involvements result in frank palsies is not borne out by anatomic evidence, only vascular lesions being found in the few instances where adequate study has been made of the fresh lesions.

Frequently, many cases of obscure hemiplegia in childhood have been attributed to encephalitis. Strümpell's polioencephalitis, exceedingly rarely, and the more frequent hemiplegic type of epidemic encephalitis do occur, but Ford and Shaffer,[23] following an extensive study of the protocols of 70 hemiplegics from 50,000 case records of the Harriet Lane Home in Baltimore, conclude that it seems possible to state definitely that the infantile hemiplegias that are associated with the infectious diseases are due to vascular lesions. Those which occur earlier in the illness are in the majority due to invasion of the blood stream by the primary organism or by some secondary invader. The palsies that develop during the convalescence are the result of vascular lesions. These complications are common to all severe infections and are not in any sense specific, although apparently specific changes in the nervous system may be produced by variola, vaccinia, varicella, and perhaps by measles and mumps. Final conclusions cannot yet be reached regarding the cause of hemiplegias in apparently healthy children. The obvious assumption of encephalitis as the cause is not supported by anatomic evidence. Only vascular lesions have been found in the few cases in which the fresh lesions have been studied. Vascular lesions may be due to invasion of the blood stream by pyogenic organisms from clinically latent foci of infection. Congenital defect in the cerebral arteries may cause hemorrhage in some. Many cortical and brain stem syndromes, acute and cerebellar ataxias and some cases of myelitis bear the same relation to the acute infectious diseases of childhood as the hemiplegias discussed. Dependable anatomic studies are still quite inadequate in these conditions.

Invasion of the blood stream by pyogenic organisms may be causative of vascular lesions, as is obvious where embolic occlusion has occurred, but infections may simply act as contributory factors in their production by general depletion, impoverishment and dehydration of the patient. Meningitis—otitic, meningococcic, tubercular and other types, obvious or unrecognized—may erode a vessel with resulting cerebral hemorrhage or cause embolic or thrombotic occlusion. During acute infectious diseases vessel wall involvements probably occur much more frequently than realized with the increased liability to actual occlusion or rupture at some later date when apparently inadequate stress or illness may serve as a final causative factor in their production. The effect of the various viruses upon the blood vessels has not been elucidated but it seems very probable that viruses and chemical agents as well as infections may involve the most terminal vascular branchings by embolic

chemicophysiologic processes which coalesce with resultant cerebral softenings or minute petechial hemorrhagic lesions.

Blood dyscrasias, particularly hemophilia and the leucemias, diabetes, congenital heart conditions, abnormalities in the biliary tract and icterus gravidus neonatorum are known to result in cerebral hemorrhage.

The comparative incidence of contributory factors* in the production of vascular encephalopathy as delineated in our study of 1,000 cases is practically reversed in young patients, arteriosclerosis usually being the most frequent etiological cause and by far the least frequent, although rarely definitely present, in young individuals. Malignant hypertensive disease was causative of cerebral hemorrhage in 7.6 per cent of the cases (32 of the 418 patients harboring hemorrhagic vascular lesions), but one of them being in the age group here considered. Hypertension is known to occur occasionally in quite youthful individuals.

Amberg[24] in a discussion of hypertension in the young, divided arteriosclerosis into three main groups: (1) those which result from kidney involvement dependent on some previous infection; (2) those that result from persistent hypertension, the cause of which is unknown; (3) those which are the direct result of infection or intoxication.

Winkleman and Echol[25] reported eight cases illustrating conditions in which cerebral vascular changes simulating arteriosclerosis have been found in childhood. Well-developed arteriosclerotic vessels were described in these fatal cases and the authors believe the conclusion is warranted that in infections and toxemias, similar less marked changes occur which may be starting points for the further development of vessel pathology. One clinical case illustrated gross damage to the vessels which resulted during a mild upper respiratory tract infection with the occurrence of convulsive seizures. Encephalographic studies showed softening in the calcarine area to account for contralateral hemianopsia. They conclude that the prevention of the acute infectious diseases of childhood will probably help to prevent many early vascular conditions.

Levin[26] reported an infant with convulsive seizures beginning at the age of four months and with hemiplegia appearing at the age of nine months. Death occurred in the ninth month from circulatory and respiratory collapse after trephining the skull. At pathologic examination of the brain multiple laminar softenings were found throughout the cerebral cortex, most marked in the parietal regions on each side. The pial arteries showed calcification of the media; intimal proliferation was present in these vessels and in the large basal arteries. In the areas of softening there were a marked proliferation of the capillaries and an abundance of phagocytic cells containing fat. About the vessels were accumulations of cells from which graduations could be traced to the fat-laden phagocytes. The condition appeared to represent an early

*Presented at the annual session of the California Medical Association at Pasadena, Calif., May 11, 1938.

TABLE I

AGES OF PATIENTS WITH VARIOUS TYPES OF CEREBRAL VASCULAR LESIONS

(Exclusive of Sinus Thrombosis, 88)

		UNDER 10	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90 PLUS	
Embolus	(133)	4	6	16	17	32	23	23	10	2		(40% 30-49)
Thrombosis	(361)	5	1	2	9	20	70	111	115	26	2	(70% over 60 years)
Hemorrhage	(418)	16	8	27	43	68	97	84	62	11	2	(38.7% under 50 and 62% less than 60 years)
	912	25	15	45	69	120	190	218	187	39	4	

stage in the development of diffuse or lobar cerebral sclerosis, a common lesion in cases of infantile cerebral paralysis. The lesions seem to be due to alterations in the circulation of the cerebral cortex. Actual vascular occlusion is rarely found and it is believed vasomotor disturbances may play an important role in the pathogenesis of encephalomalacia in infancy.

A STUDY OF FORTY INSTANCES OF CEREBRAL VASCULAR INVOLVEMENT IN YOUTHFUL SUBJECTS AMONG 1,000 NECROPSIED CASES OF VASCULAR ENCEPHALOPATHY

In a study of 1,000 necropsied cases with cerebral vascular involvement found at 12,503 consecutive autopsies done at the Los Angeles General Hospital, the age incidence of the patients was given special attention. It was found in this series, exclusive of 88 cases of sinus thrombosis,* that 25 of the patients were under 10 years of age, and 15 were between 10 and 19 years of age. The average age of the patients with sinus thrombosis was 25 years with 26 of the 88 being under 10 years, and 12, 10 to 19 years of age inclusively. This usual generally low age incidence and the most common etiological factor of infections of the face and otic apparatus in sinus thrombosis excluded this type of intracranial involvement from a study of distinct cerebral vascular lesions in the young.

Of the 133 patients demonstrating embolic processes, 4 were 9 and under and 6 were 10 to 19 years old. Thrombotic lesions were found in 361 of the 1000, five being under 10 years of age and one was 17 years old. Five of these young patients demonstrated venous thromboses and a 2-year-old male had thromboses of the internal carotid arteries bilaterally. Sixteen of 418 patients with cerebral hemorrhage were 9 and under, while 8 were 10 to 19 years of age. Thus 4.4 per cent of 912 patients with various types of cerebral vascular lesions were under 20 years of age, a low age incidence in patients with vascular encephalopathy, not generally recognized as existing. The age groups of all the patients in this survey with the various types of lesions are given in Table I.

YOUTHFUL PATIENTS WITH EMBOLIC CEREBRAL PROCESSES

The pathologic findings in these 10 young patients were similar to those in older cases but usually there was but a short duration of an infectious illness without the usual clinical signs of endocarditis where such involvement was found at necropsy. The pathologic etiology in 6 of these 10 young patients in whom embolic cerebral process had developed was endocarditis, in 2, otitis media was the infectious source, while in one each it had been empyema and osteomyelitis of the clavicle. Their ages were 10 months and 5, 7, and 8 years, with two, 16 years

*Reported in Ann. Otol. Rhin. & Laryng., March, June, and September, 47: Pages 78, 402, 411, 775, 1938.

old; one, 17 years, and 4, 19 years old. The cerebral vascular involvements demonstrated were embolic processes in the cortical branches of the right middle cerebral artery in two, bilateral middle cerebral cortical branch emboli in two, bilateral middle cerebral cortical emboli and softening in the distribution of the left posterior cerebral artery in one and embolic processes in the cortical branches of the right middle cerebral artery with thrombosis of the superior longitudinal sinus in the 10-month-old babe. The left middle cerebral artery contained an embolic occlusion which extended into the ophthalmic artery in a 5-year-old child. Petechial cortical involvement was shown in an 8-year-old female whose left ear had discharged intermittently for several months. Multiple cerebral abscesses were found in the presence of bilateral empyema with pulmonary abscesses in a 19-year-old male and subdural hemorrhage appeared in a 7-year-old male with subacute endocarditis.

The cerebral lesions in these young individuals were generally less marked and extensive than in older patients, probably due to the usual shorter survival period following cerebral involvement. The primary branchings of the major cerebral vessels were also relatively less frequently occluded, 50 patients of the entire group of 133 showing complete solitary embolic occlusions of major vessels and 12 more with multiple lesions demonstrating major vascular occlusions, while in these youthful individuals but one displayed a major cerebral arterial thrombosis.

A number of illustrative cases available from this series studied, demonstrating the various contributory factors in the production of cerebral vascular pathology in these young individuals, have had to be omitted because of space limitation.

CEREBRAL THROMBOTIC LESIONS IN THE YOUNG

Among 361 patients demonstrating thrombotic cerebral involvement at necropsy but 5 were under 10 years old and one was 17 years of age.

It is apparent, as evidenced in this survey, that thrombotic cerebral processes in young patients are of quite low incidence and that cerebral arterial occlusions are definitely uncommon, but one instance of a cerebral artery thrombosis appearing in a patient under 20 years of age among these 1,000 consecutive necropsied cases with vascular encephalopathy. In the other 5 youthful patients with thrombotic lesions the cerebral veins had been involved. These 5 constituted one-half of the incidence of cerebral venous thrombosis* found in the entire survey. The comparative incidence of embolic cerebral processes found in the young was 7.5 per cent (10 of 133 cases); of thrombotic lesions 1.66 per cent (6 of 361), with but 0.3 per cent with arterial thrombosis; and of hemorrhagic cerebral involvement 5.74 per cent (24 of 418).

*Venous Thrombosis, Ann. Otol. Rhin. & Laryng. 47: 775, 1938.

CEREBRAL HEMORRHAGE IN THE YOUNG

The ages of the 24 youthful patients suffering fatal cerebral hemorrhage were 1 hour; 4 hours; 2, 4 days; 13 days; 1 month; 4, 2 months; 2, 5 months; 22 months; 2, 7 years; 8 years; 2, 12 years; 13, 14, 16, 17, and 2, 18 years; 16 being 8 years of age and under and 8 were between 12 and 18 years of age. Four males and 4 females were in the second decade of life while 11 males and 5 females were under 9 years of age.

Etiologic contributory or causative factors in the resultant hemorrhages were of a congenital nature, due to impaired development, in a 7-year-old patient with Little's disease; in 2 infants 4 days old displaying icterus gravidus neonatorum and in one, aged 2 months, with congenital absence of the extrahepatic bile ducts and gall bladder. One 5-month-old baby had a large thymus. Infections were contributory in 2 babies, dying 1 and 4 hours respectively following birth, from subarachnoid and subdural hemorrhages of syphilitic cerebral vessels. Pial arachnoid hemorrhage appeared in the presence of tubercular meningitis in a 7-year-old male. A perforating vessel displaying tubercular arteritis was traced to a hemorrhage in the basal ganglia in a 5-month-old baby. Multiple cerebral hemorrhagic lesions were found in a baby of 2 months with bronchopneumonia. Subdural hemorrhage was found in a boy 13 years of age with typhoid fever, and a massive, ruptured, intracerebral hemorrhage appeared in a 17-year-old boy demonstrating splenic enlargement. A boy of 12 years, developing poliomyelitis, died from intraventricular hemorrhage. Acute arthritis was present in a 16-year-old girl with subarachnoid hemorrhage, as was also displayed by an 18-year-old eclamptic. Subarachnoid hemorrhage appeared in a 2½-month-old male with whooping cough. An 8-year-old diabetic boy died from ventricular hemorrhage. The diagnosis of Van Jaksch's disease was confirmed at necropsy in a 2-month-old girl suffering from ventricular hemorrhage. Acute leucemia was present in a boy of 12 years displaying a clot in the inferior parietal lobule. Marked sclerosis of the vessels of the circle of Willis with cystic degeneration in the entire left parietal lobe was shown in the brain of an 18-year-old girl who had suffered a sudden hemiplegia one and one-half years before she was admitted to the hospital with a blood pressure of 270/110. No satisfactory causes were discovered for the subdural hemorrhage found in a 1-month-old baby, the bulbar hemorrhage in a 22-month-old child, the hemorrhage in the basal ganglia of a 13-day-old baby or the cerebellar hemorrhage in a 14-year-old girl.

The sites and types of hemorrhagic encephalopathy shown by this group were subarachnoid in 6, subdural in 4, in the basal ganglia in 5, ventricular in 3, petechial and multiple in 2 each, and bulbar and cerebellar in 1 each.

SUMMARY

The incidence and pathology of cerebral injuries in the newborn infant have received much consideration, but the relative frequency of spontaneous cerebral vascular lesions is not generally realized. The recognized causes of vascular encephalopathy in the young have been recited and opinions regarding the mechanisms of the pathologic developments are presented.

A study of 40 instances of cerebral vascular involvement in youthful patients is reported, showing the low age incidence, the etiological factors concerned, and the types and sites of the pathology. Embolic cerebral involvement appeared in 10, thrombotic in 6, with the arterial system being involved in but one, and hemorrhagic lesions were present in 24 patients, under 20 years of age.

The recognition of the potentiality of cerebral vascular involvements in quite young individuals, harboring conditions shown to be contributory to such pathology, increases the efforts in treatment of the initial processes in the attempt to avoid such serious complications.

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THE INTRADERMAL SALINE TEST (McCLURE-ALDRICH) IN THE NEWBORN INFANT

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IN 1923 McClure and Aldrich[6] described a test, now known by their names, which they believed was significant in edematous states. This test consisted of producing a wheal in the skin, preferably of the forearm, by the intracutaneous injection of 0.2 c.c. of an 0.8 per cent aqueous solution of sodium chloride. The rapidity with which the wheal disappeared was in direct ratio to the amount of edema. The test was also used in patients as an indication of the tendency of edema to increase or diminish, for it was found that the disappearance time decreased with, or preceding, development of edema, and increased with, or before, subsidence of edema. In normal adults McClure and Aldrich found the disappearance time to be from 60 to 80 minutes; in children over 1 year of age it was more than 60 minutes.

During the course of a study conducted in 1937 on identical twins, one having anemia of the newborn and the other having universal edema of the newborn,[9] it was of interest to note the increasing disappearance time of the intracutaneous wheal as recovery took place. The present study was undertaken to furnish for comparison the results obtained in normal infants during the newborn period.

In all, 60 infants were tested, 32 boys and 28 girls. They were all born at full term, normal in all respects. The birth weights ranged from 5 pounds 5 ounces to 9 pounds 1 ounce, with an average of 7 pounds 1 ounce. At the time the tests were performed, the infants' ages were from 1 to 9 days (Table I), and the weights ranged from 5 pounds 2 ounces to 8 pounds 15 ounces, with an average of 7 pounds.

TABLE I

AVERAGE DISAPPEARANCE TIME IN MINUTES

AGE IN DAYS	NUMBER OF INFANTS	AVERAGE TIME	AVERAGE TIME FOR 3-DAY PERIODS
1	6	20.5	
2	4	23.0	
3	12	19.0	20.8
4	11	19.5	
5	9	20.0	
6	4	23.5	21.0
7	4	18.5	
8	6	19.5	
9	4	23.0	20.3

The average disappearance time for the group was 20.7 minutes. No significant difference was noted between the sexes, the disappearance time averaging 20.9 minutes in males and 20.5 minutes in females.

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The question of whether the physiologic loss of weight causes a more rapid disappearance of the wheal[4] was considered. I found that it does not, for many infants whose weight at the time of the test was below the birth weight showed a longer disappearance time than some who were already heavier than at birth. And, as may be noted in Table I, there was no constant trend found according to age, either when considered daily or in intervals of three-day periods.

The time of disappearance of the wheal varied between 13 and 30 minutes, with more than two-thirds of the infants ranging between 17 and 24 minutes (Chart 1).

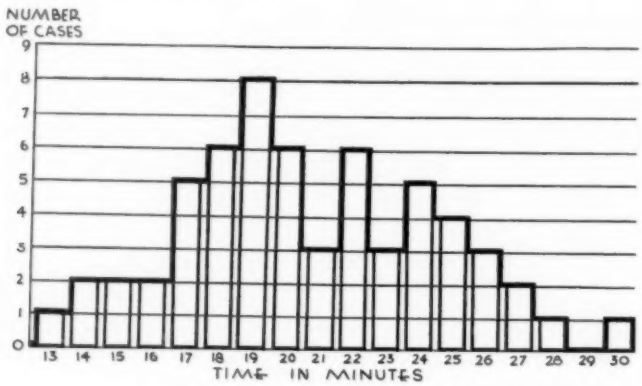

Chart 1.—Disappearance time in 60 newborn infants.

DISCUSSION

The literature on the intradermal saline test in the neonatal period is scant. There is no question that from infancy to adulthood the disappearance time increases in length,[2-5, 7, 8] with actual figures of various authors rarely concurring. But are there daily significant changes immediately after birth? My findings do not indicate this to be so. Lelong and Pétrovich,[4] on the other hand, seem to have found otherwise. They reported an accelerated rate concomitant with the physiologic drop in weight. Moreover, their figures in general seem to be lower than those of other investigators. In 19 cases they reported a disappearance time of 11 minutes at 1 week; and in 15 cases, there was a disappearance time of 17 minutes at 2 weeks. On the basis of comparatively few tests, others[1, 2, 10] have reported figures for this early period ranging from 11.5 to 40 minutes.

SUMMARY AND CONCLUSIONS

1. The intradermal saline test (McClure-Aldrich) was performed on 60 normal infants from 1 to 9 days old. There were 32 boys and 28 girls.

2. The average disappearance time for the group was 20.7 minutes, with no significant difference between the sexes; it was 20.9 minutes for the boys, and 20.5 minutes for the girls.

3. No constant trend was found according to the age or weight of an infant during the first 9 days of life.

4. The disappearance time varied between 13 and 30 minutes, with more than two-thirds of the infants ranging between 17 and 24 minutes.

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PROPHYLAXIS AGAINST WHOOPING COUGH IN EXPOSED CHILDREN, WITH SPECIAL REFERENCE TO SERUM

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PROGRESS in prophylaxis against whooping cough among children who have not been exposed to the disease has been encouraging in recent years.[1-4] In such prophylaxis we are anticipating an event which may never occur or may not occur for years: that a given child should be in close contact with an active case of whooping cough. Still generally unsolved and much more distressing is the problem of prophylaxis in a child, particularly an infant, who has just been intimately exposed to an early case of whooping cough and who has a history of no previous protection. In an effort to overcome this predicament, we have in our whooping cough clinic tried different therapeutic methods and have studied their effects in an attempt to prevent or modify a disease which is very grave in infants. In 1,000 cases of whooping cough 101 contacts availed themselves of some form of preventive inoculation. Of this number we could carefully follow up the course of 88 children.

No child was treated unless he had been definitely and intimately exposed to an active case of pertussis and had had no previous prophylactic vaccine therapy, such as the Sauer vaccine or its equivalent. In addition, since the exposed cases were almost entirely familial, the exposure to the disease continued, for in clinic patients it was impossible to segregate the children, as the homes were too crowded. This is a factor which allows for the maximum degree of contagion and a high incidence of communicability which, according to different authors, varies from 70 to 100 per cent.[1, 2] We felt that any procedure demonstrably effective under such circumstances would leave no doubt of its usefulness. The different prophylactic agents selected for trial were Sauer's pertussis vaccine, Krueger's undenatured bacterial antigen (U.B.A.), Mulford's topagen (soluble) injected hypodermically, adult blood serum, hyperimmune serum, and finally convalescent human serum.

We tried Sauer's vaccine for a small group although we had regularly observed it to be useless in the face of exposure when given in the usual manner at weekly intervals.[4] We felt, however, that there was a bare possibility of this vaccine's proving of value in children actively exposed to whooping cough if it was given more intensively and with greater frequency. Accordingly, 10 children, all of whom had intimate exposure in an environment with siblings suffering from the

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disease, received daily injections of Sauer's vaccine for seven days, in a dosage of 1 c.c. daily, making a total of seventy billions of bacteria injected in each individual. Of the 10 children so treated, 7 contracted the disease and 3 did not. In several pertussis developed before the week had elapsed. The nature of the disease developed in the course of this intensive vaccination seemed to be in no way unusual and was no milder than the expected course (Table I, line 1).

The second group of 10 children, all intimately exposed, received Krueger's undenatured bacterial antigen, mechanically derived from pertussis bacilli, in doses of 1 c.c. every other day for a week. Here also, 7 contracted the disease, running a course not discernibly different from the average case of untreated whooping cough which we were seeing in the clinic by the hundreds. It is interesting to note that in both vaccine-treated groups the percentage contracting the disease was 70 per cent, the usual incidence of pertussis in children intimately exposed to the disease (Table I, line 2).

The third contact group of ten children, all intimately exposed, received three injections every other day of 1 c.c. of Mulford's topagen (soluble). This is a soluble antigen derived from the supernatant fluid of pertussis bacilli which have been mechanically triturated. While this preparation had been used as a form of intranasal therapy, it had been previously used by hypodermic injection in only two cases.[5] Our dosage was a matter of conjecture as we had little precedent to follow. Out of 10 children so treated, 6 contracted an average form of the disease and 4 escaped. Since the results were not very promising, we proceeded no further with this method (Table I, line 3).

The fourth contact group was composed of 13 children, 12 of whom had intimate contacts and 1 extrafamilial contact. They received intramuscular inoculations of parental serum prepared only from parents who gave a definite history of having had pertussis and who were re-exposed without symptoms. Twenty or 40 c.c. of such serum was injected—the smaller dose for infants and the larger dose for older children. Eight children completely escaped the disease and the remaining 5 had courses distinctly milder than the vaccine-treated patients or our untreated control patients (Table I, line 4).

Our criteria of the severity of a case are (1) number of paroxysms, (2) amount of vomiting, (3) loss of weight, (4) freedom from complications, and (5) duration of the disease.

These were carefully recorded by mothers in all our cases, and these data, studiously surveyed by one of us, with corroborative questioning. Most of these charts were kept in a manner which reflected credit upon the carefulness and intelligence of these mothers. In fact, this entire work was carried on with admirable cooperation by the parents. For this reason we felt we could judge objectively the results of our specific therapy and not depend upon the mere opinion of a lay

person. We learned, moreover, the mother's nocturnal waking hours were an excellent index of the severity of a case. The better the mother slept, the better the progress of the child with the whooping cough.

This work was begun four years ago as a treatment clinic for active cases of whooping cough. At the very outset we were confronted by contacts who were compelled to remain in the environment of contagion. In an effort to find an effective agent in the face of active and continued exposure, we tried, as noted above, different measures in an attempt to discover a prophylactic measure, reliable, safe, cheap, and easily available. Finding the easily procured vaccine ineffective, we turned to the use of serum obtained from adults who gave the history of an attack of whooping cough. We considered the results with adult human serum good, but we conceived the idea that we might improve our results by increasing the immunity of such adults. We thought this might be accomplished by inoculating these volunteers with the Sauer or the Mishulow vaccine in very large doses. Only after we had been doing this work for some time did we learn that Kendrick,[7] and before her Jundell,[8] had performed the same experiment on a small scale. To the serum thus obtained, Kendrick gave the name of hyperimmune serum.

We procured our serum from male adults who gave a definite history of having had whooping cough, whose Wassermann tests were negative, and whose examinations revealed freedom from disease. Over a period of from six to eight weeks 20 c.c. of Sauer's or Mishulow's toxin vaccine were injected. Phlebotomies were performed one and two weeks later, and the serum was separated from the blood so obtained. The serum was injected intramuscularly into 12 contact children, all with familial exposures, in dosages of 10 c.c. in infants and 20 c.c. in older children. Of these 12 subjects, 8 were completely protected, and 4 were not. The four who contracted pertussis ran mild courses, distinctly less severe than our numerous controls, using the same criteria of severity noted above (Table I, line 5).

During all this time we were collecting blood from our patients who were recovering from pertussis. Since six weeks after the disease has come to a standstill is believed to be the period of highest immunity,[6, 9, 10] the bleedings were performed at this time. If complement fixation tests can be used as any guide to the height of the immune titer, these convalescent patients had attained a high degree of immunity, as their complement fixation tests were markedly positive. Thus a sixth group, our largest, we studied for the effects of this convalescent serum upon children in contact with active whooping cough. At the beginning we used from 5 to 10 c.c., according to age, but in a short time increased our dose to from 15 to 40 c.c. Infants were injected with at least 15 c.c. and the older children with 40 c.c. With

these larger doses our results were striking. Of the 33 children so treated, of whom 30 had intimate contact with the disease, only 5 contracted the disease, and these ran extremely mild courses. Our highest percentage of prevention, 85 per cent of the children intimately exposed, was attained through the administration of convalescent serum (Table I, line 6). These results were attained to a large extent by our policy of injecting every child exposed to coughing children even when these were only suspicious of whooping cough. Occasionally a child was thus injected who was not exposed to whooping cough, but these are not included in our statistics. By the early administration of large doses of convalescent serum we achieved results superior to those obtained by the administration of 10 to 20 c.c. of hyperimmune serum. It is quite possible that by increasing the dose of hyperimmune serum we can equal the percentage of prevention obtained with convalescent serum. This is now being studied.

TABLE I

METHOD OF PROTECTION	NO. OF CONTACTS		TOTAL NO. OF CONTACTS	DOSAGE	NO. OF FAILURES	PROTECTION PER CENT	COURSE OF FAILURES
	FAMILIAL	EXTRA-FAMILIAL					
Sauer vaccine	10	0	10	80 bil.	7	30	Normal
U. B. A.	10	0	10	40 bil.	7	30	Normal
Topagen (soluble)	10	0	10	3 c.c.	6	40	Normal
Adult blood serum	12	1	13	20-40 c.c.	5	61	Mild
Hyperimmune serum	12	0	12	10-20 c.c.	4	67	Mild
Convalescent serum	30	3	33	15-40 c.c.	5	85	Very mild
Total	84	4	88				

It was our impression that the best results with the different sera used were obtained when larger dosages were given. Furthermore, in a disease like pertussis, which is virtually impossible to diagnose in the early stages, a history of exposure or a strong suspicion of whooping cough in an afebrile child with a negative examination should lead to protection of the exposed children in the same family. If this is not done, then many, many children will be inoculated at the very end of the incubation period or in the early stages of the disease. This is because it takes close to two weeks to diagnose an unsuspected case of whooping cough. The blood count is not positive before this time.[11] Only the cough plate has proved of diagnostic value in the early stages, and this can only be invoked when the disease is suspected. Instead of losing three to five days awaiting the result of cough plates, we felt it wiser to inoculate children exposed to suspected whooping cough cases.

With this procedure, the 5 children who had received convalescent serum but developed whooping cough ran such a mild course that under ordinary circumstances the diagnosis would have been difficult.

Analysis of our failures showed that they were due either to too small a dose of serum or to injection too late in the incubation period. When the disease occurred shortly after the injection, we felt that the latter must have been the reason. While it may be a process of rationalization, we have the impression that if we could inoculate an exposed child sufficiently early and with the maximum dosage, we could obtain even better protection.

DISCUSSION

In 1935 Bradford[12] reviewed the literature on the use of convalescent serum and adult blood for the prevention of whooping cough. His review is so thorough that any reader interested is referred to his article for details. Such a summary, however, reveals that up to 1935 there had been no large series of cases treated with large doses of serum. It is noteworthy that practically all previous authors seemed to feel that even with the dose they used, usually about 10 c.c., the results were good. In Bradford's series of 44 cases of intimate exposure, 17 children were given vaccine therapy with no modification, with results similar to ours. The other 27 cases were given 20 c.c. of whole blood or 10 c.c. of convalescent serum prepared during the eighth week of convalescence from whooping cough. Bradford did not distinguish between those who received serum and those who received blood but reported that 15 of the 27 cases injected developed pertussis (56 per cent). We believe that this figure could have been definitely improved by administering larger doses, comparable to ours. Of the 15 children who contracted the disease, 10 ran courses so mild as to indicate modification. Of 20 untreated controls, under the same circumstances, all contracted pertussis. In a later report (1936) Bradford states that 25 more cases treated similarly gave the same results.[13]

Since Bradford's work, Meader[14] treated 115 exposed children with convalescent serum. This is the largest series on record, but the degree of intimacy of exposures was not described. With the administration of 10 c.c. of convalescent serum to his contact cases, 37 (32.2 per cent) developed whooping cough. Since 8 children coughed the day after inoculation, these failures can be omitted. Allowing for this correction, 29 of 107 (27 per cent) exposed children developed cough after receiving only 10 c.c. of convalescent serum. Of 183 control subjects, 121 (68 per cent) developed whooping cough—a striking difference in incidence from those who had received the benefit of convalescent serum. Good as these results are, we feel that they could have been surpassed by at least doubling the dosage of serum. On analysis it is clear that some patients applied for treatment late in the incubation period, and in our hands they would have been injected with 40 c.c. of serum. The increased dosage of serum in the late incubation stages is analogous to the use of convalescent

serum in measles, in which the dosage to be fully effective should be increased in proportion to the number of days of exposure.

Schermerhorn, in 1938, reported on the Grand Rapids pertussis immunization study.[2] In this study convalescent serum was injected in intimately exposed children in doses of 20 c.c., and one week later another 10 c.c. intramuscularly. In a group of 30 patients so treated, 8 (27 per cent) developed pertussis, while 29 controls showed a whooping cough incidence of 82 per cent. Their convalescent serum was collected from donors as late as one year after the attack of pertussis. In light of this, it seems that this convalescent serum was not as potent as our convalescent serum, which was collected six to eight weeks after the disease was over, or at the height of immune process. Thus, while their dosage seems adequate for most cases, the use of serum of lower potency should call for a larger dosage. If this were done, their excellent results might have been further improved.

In Grand Rapids vaccine therapy tried as a prophylactic agent in children actively exposed to whooping cough gave protection in 18 out of 27 patients. This is a far better record than our results, 67 per cent protection as against 30 per cent. The Grand Rapids technique was much the same as the one we employed. From 1 to 1.5 c.c. of Sauer's vaccine was injected daily into exposed children until a total of 8 c.c. had been given. We cannot explain the superior results in Grand Rapids in the light of our failure, as they are contrary to others reported in the literature.[1, 12]

With regard to what Kendrick has termed hyperimmune serum, our prophylactic results are similar. Kendrick obtained her serum from adults who gave a definite history of having had whooping cough. From 7 to 20 c.c. of vaccine distributed over six to ten weeks was then injected into these adults. Ten days after the last injection, the blood was collected and the serum separated. The serum thus processed showed a high titer of agglutinins for *B. pertussis* and gave a very strong opsonic reaction.

The effect of this serum was studied in 22 babies, all directly exposed to active whooping cough; 10 c.c. was injected intramuscularly, and one week later the same dosage was repeated. Of 11 babies inoculated before symptoms appeared, 9 gained complete protection, and the 2 babies who contracted whooping cough ran distinctly mild courses. Six babies given the same serum after symptoms appeared showed no apparent modification of the disease. Four of 5 control subjects developed pertussis, of which 3 were severe cases. The Grand Rapids workers thus arrived at the opinion that a serum completely protecting 9 of 11 babies actively exposed to whooping cough and modifying the cases in the remaining 2 was very worth while and useful. We were able, with

a similar serum, to protect completely 8 of 12 exposed children and to modify the disease in the other 4. We believe it is good practice to give another 10 c.c. intramuscularly five to seven days after the first injection, and we shall pursue that practice in the future. Hyperimmune serum is more easily available and is available in larger quantities because the source is vaccinated adults. Large quantities of serum can be obtained, and, if this is shown to be as effective as convalescent serum (which it may be in equivalent dosage), the problem of serum supply is solved.

Bradford[13, 15] and Singer-Brooks[16] have recently shown that convalescent whooping cough serum increased the phagocytosis of *B. pertussis*. Thus it seems that, as in the case of sulfanilamide, there is no direct bactericidal effect, but the opsono-cytophagic power of the leucocytes is increased. The same holds true for serum of children previously injected with *H. pertussis* antigens.

CONCLUSIONS

1. Over 100 children directly exposed to whooping cough were treated with various prophylactic measures.

2. Intensive daily therapy with Sauer's vaccine had no noticeable effect on the incidence or course of whooping cough in exposed cases.

3. Krueger's vaccine was similarly ineffective in cases exposed to whooping cough.

4. Topagen (Mulford, soluble) had no apparent effect, given in 3 c.c. doses, upon the incidence or course of whooping cough in intimately exposed children.

5. Adult blood serum was effective in 61 per cent of our subjects intimately exposed to whooping cough. The course of those who contracted the disease after injection was generally modified. The doses recommended are 20 c.c. for infants and 40 c.c. for older children.

6. Hyperimmune serum can be obtained from an individual with a history of having had whooping cough after he has been inoculated with large doses of vaccine (20 c.c.) over a period of six to eight weeks. Serum is obtained one or two weeks later. Such serum protected two-thirds of our cases, and modified the rest, though intimately exposed to the disease.

7. Convalescent serum was found to be the most effective prophylactic agent in children exposed to whooping cough. We used and recommend much larger doses than previously reported. We advise 15 c.c. to 20 c.c. for an infant and 40 c.c. for an older child.

8. Analysis of the failures with the use of serum shows either a dosage which was too small or injection too late in the incubation period.

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THE TREATMENT OF GONORRHEAL VULVOVAGINITIS IN CHILDREN WITH SILVER PICRATE SUPPOSITORIES

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INTRODUCTION

ALTHOUGH scores of therapeutic agents have been used in the treatment of gonorrheal vulvovaginitis, there is no one which is fully satisfactory. Estrogenic hormone in suppository form is probably most effective,[1] but, due to the high cost of this therapy, its use in many clinics is not practicable. In addition, the side effects of the hormone in producing temporary breast enlargement and pubic hair in some patients would make it desirable to use a purely local agent if an effective one could be found.

Survey of the records of patients admitted over a period of five years to the Philadelphia General Hospital with gonorrheal vulvovaginitis shows rather discouraging results of therapy. In a series of 25 patients treated by vaginal instillation of silver nitrate in boric acid ointment, an average of 7.3 weeks was required to obtain the first of three consecutive negative smears. With other agents the periods required were somewhat shorter, examples being: sulfanilamide (42 patients) 5.5 weeks;[2] progynon parenterally (25 patients), 3.2 weeks; floraquin suppositories (10 patients), 1.4 weeks. Complete data on recurrences were not available, but it is known that the latter treatment was discontinued because of recurrences in 6 of the 10 cases.

Lewis and Adler[3] reported 33 cases treated with estrogenic suppositories. An average of 2.9 weeks was required for obtaining negative smears, and 5 cases were listed as recurrent. Reichert and coworkers,[4] in a clinical study of various local antiseptics, including mercurochrome, merthiolate, strong silver protein, metaphen, and lactic acid, reached the conclusion that the silver salts were the most efficacious of these in the treatment of gonorrheal vaginitis.

Recently, Kobak and Frankenthal[5] introduced the use of silver picrate suppositories in children with vulvovaginitis. They chose the suppository form of administration because of the ease of application, accessibility to the crypts of the vaginal mucosa, and prevention of premature expulsion. In view of their encouraging results in a series of 13 cases of gonorrheal vulvovaginitis, we considered that such a form of therapy, which would be less expensive than hormone administration, was worth further trial. Consequently such a study was begun in 1937 in the Children's Clinic and Ward of the Philadelphia General Hospital.

From the Pediatric Department of the Philadelphia General Hospital.

PLAN OF STUDY

The cases studied included children up to twelve years of age in whom a diagnosis of gonorrheal vulvovaginitis had been made on the basis of vaginal discharge with one or more vaginal smears showing the presence of gram-negative intracellular diplococci.

Both acute and chronic cases were included, some having applied for treatment a few days after the onset of the vaginal discharge, others having been treated unsuccessfully for periods of weeks or months. The silver picrate suppositories used consisted of 1 grain of silver picrate incorporated in a boroglyceride base, molded in the usual "bullet" suppository form.*

On the initial clinic visit the mother was instructed in hygienic measures, including attention to the patient's general physical condition and to the local discharge, for which external cleansing of the vulva with soap and water was recommended. The method of inserting the silver picrate suppository was then demonstrated to her, using a finger cot on the index finger. She was instructed to introduce one suppository each night at bedtime with the exception of the night immediately preceding the weekly clinic visit. This suppository was omitted in order to make the vaginal smears more reliable, as silver picrate was often noted in the vagina as long as twelve to eighteen hours after introduction. If difficulty in inserting the suppositories was reported, a nurse visited the home to give assistance, although this was found necessary in three cases only.

At each visit to the clinic the presence or absence of vaginal discharge was noted, and a smear was taken from deep in the vagina. Treatment was continued until three consecutive negative smears, with no discharge, were obtained. During the period of treatment the children were observed weekly, being told subsequently to return at monthly intervals for checkup by clinical examination and vaginal smear. Routine vaginoscopic examination was felt to be impractical in a large clinic and was not, therefore, made routinely. In the few cases so examined, the findings did not help us in deciding whether treatment should be continued.

The silver picrate was used only in colored children because of the possibility, however remote, of producing argyria. Each suppository containing 1 grain of silver picrate was approximately equivalent in silver content to 1 c.c. of a 10 per cent solution of mild silver protein, or to 3 c.c. of a 1 per cent solution of silver nitrate. No pigmentation of the gums or mucous membranes was noted in these children.

RESULTS

A total of 30 patients with gonorrheal vulvovaginitis were given the silver picrate suppositories over periods of from three to twenty-

*We are grateful to John Wyeth & Brother, Inc., for supplying the silver picrate suppositories.

nine weeks (Chart 1). Of these 30 cases, 16 were classified as chronic with histories of discharge for periods over one month; 11 were classified as acute; and 3 cases came with no history of duration of discharge.

Treatment was continued in all cases until the children had been free of discharge, with no gram-negative diplococci showing in vaginal smears, for three consecutive weeks. In 5 cases it was necessary to reinstitute treatment because of recurrence. In 1 case treatment was stopped temporarily because the child was critically ill with an obstructive laryngitis.

Of the 30 patients, 20 were followed for at least six months, 5 for at least four months. The remaining 5 patients followed for periods of from one to four months failed to return for later check up and could not be located because of change of address.

Of the total 30 patients, 18 had their first negative smears at the end of the first week, 23 having become negative by the end of the second week. Of the 14 patients with profuse discharge at the beginning of treatment, 9 showed a definite decrease in amount at the end of one week's treatment, while only 2 patients continued to have profuse discharge after two weeks of treatment. Following the cessation of profuse discharge, most cases showed slight persistent discharge for periods ranging from one to three weeks. The average period of time required to obtain the first of three negative smears was 3.2 weeks. This includes one resistant case which was treated for twenty-six weeks, and which, if excluded, would bring the average time to 2.4 weeks.

As to the incidence of recurrent and resistant cases, we felt that conclusions could be drawn only from the 20 cases followed at least six months. That these cases were representative of the entire group is evidenced by the fact that the average time for the first negative smear to be obtained was the same as for the entire group of 30 patients. Of the 20 patients adequately followed, 6 had a recurrence of either positive smear or discharge within two to six months after treatment was stopped. Of these 6, 5 had only a single recurrent positive smear with no discharge, and when treatment was reinstituted became negative within a week and remained free of demonstrable gonococci for the balance of the observation period. Although the remaining 10 patients were not followed for a full six months, 3 showed recurrences while under observation, making a total of 9 known recurrences for the 30 patients.

Several patients proved resistant to treatment. Despite regularly administered therapy over periods of from twenty-six to thirty-three weeks, 3 patients continued to have intermittently positive smears with slight persistent discharge. It was of interest to us that these 3 patients all had serious associated systemic disease (glomerulonephritis, Pott's disease, and pulmonary suppuration) upon admission

to the clinic. It was felt that this might have been significant in view of the fact that they were the only ones of the entire group who were in poor health.

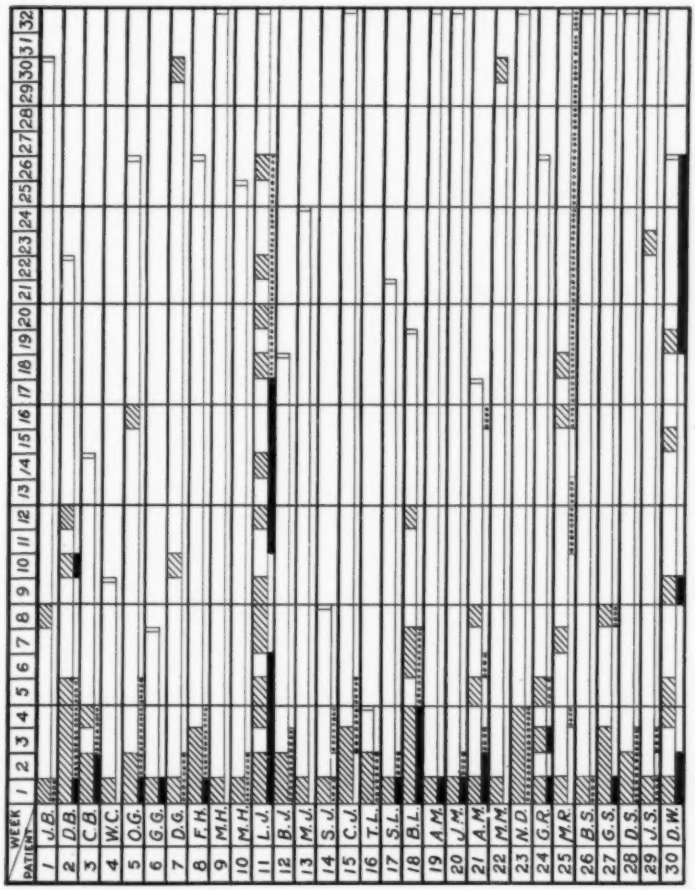

Chart 1.—Results of treatment in gonorrheal vulvovaginitis with silver picrate. For each case the upper bar represents the period of weeks for which the patient was followed. Cross hatching in this bar represents the weeks on which the vaginal smear was positive. The lower dotted bar indicates persistence of discharge, with profuse discharge indicated by solid black.
Thus, Patient 1 (J. B.) was followed thirty weeks with positive vaginal smears in the first and eighth weeks, but with discharge only during the first week.

DISCUSSION

Anyone who has observed a number of cases of gonorrheal vulvovaginitis must have realized the difficulties in judging the effectiveness of the usual therapeutic agents. One patient with an acute infection would show rapid improvement, while a similar case on the same treatment would show persistent discharge and frequent positive smears for months.

The determination of recurrence or reinfection was disturbing. We saw patients go along absolutely free of discharge only to flare up or again develop a positive smear. Questioning the mothers was seldom of help in deciding whether the child had been reinfected or had been harboring the gonococci in some focus.

Our results in 30 cases were generally in accord with those of Kobak and Frankenthal.[5] Profuse discharge cleared up rapidly, while in 18 of 30 cases negative smears were obtained after a week's treatment. This figure is much more favorable than that found by us[2] with sulfanilamide, with which only 6 cases of 42 were negative at the end of one week.

Comparison with other treatments used in the hospital previously also shows a favorable result. This is presented in Table I, which shows the number of cases rendered negative by smear at the end of one and two weeks' treatment.

TABLE I

CASES OF VULVOVAGINITIS, PEDIATRIC SERVICE PHILADELPHIA GENERAL HOSPITAL, 1932-1937

	SILVER PICRATE	SILVER NITRATE	PROGYNON	SULFANILAMIDE
Total cases	30	25	25	42
Number of cases with negative smears at end of 1 week	18	6	8	6
Number of cases with negative smears at end of 2 weeks	23	5	7	12

We felt that the silver picrate was effective in most cases in rapidly clearing up the discharge and causing gonococci to disappear from the vaginal smears. In this respect it seemed to be a better therapeutic agent than most of the commonly used antiseptics, and it compared favorably with estrogenic substance.

As to recurrence, appraisal is more difficult. The incidence in our series was higher than that reported for estrogenic suppositories.[3] While we felt that hormone therapy would have been more effective in preventing recurrences, we did not feel that the results were unduly poor viewed in the light of the frequent recurrences characteristic of the disease.

CONCLUSIONS

Thirty cases of gonorrheal vulvovaginitis treated with suppositories containing silver picrate have been presented. Most cases showed cessation of profuse discharge and disappearance of gonococci from vaginal smears within one to two weeks. The average time required for all cases was 3.2 weeks. Of twenty cases followed at least six months, six were listed as recurrent.

Silver picrate suppositories appear to be a simple and comparatively effective treatment of gonorrheal vulvovaginitis.

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PORTAL CIRRHOSIS IN CHILDHOOD

Report of Three Cases

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PORTAL or Laënnec's cirrhosis in childhood is a rather uncommon condition. As Poynton and Wyllie[1] have shown, in childhood, biliary cirrhosis is the more common type found. This is due largely to the occurrence of congenital deformities in the bile system. In reporting three cases of portal cirrhosis, it is our intention not merely to add to the total number already in the literature, but to obtain a better understanding of the pathogenesis of the disease by contrasting the early and late stages. In addition, the surgical treatment of one case may add to our knowledge of a procedure frequently performed in adults but rarely in children.

Almost all of the recent reports of cirrhosis of the liver in childhood refer to the difference of opinion as regards the incidence of the disease. Apparently the controversy has not as yet been decided. Mallory[2] found only eleven cases in the first two decades of life, among a total of 2,066 autopsies in children over a period of thirty-five years. This total includes all kinds of cirrhosis, as do most of the other statistics to be quoted. Graham Forbes[3] reported forty cases in 15,500 post-mortem examinations at Great Ormond Street Hospital over a forty-five-year period. These were predominantly in later childhood. Sir Humphrey Rolleston[4] stated that there were only twelve cases of portal cirrhosis in patients under 21 years of age among 16,100 necropsies at St. Georges Hospital from 1865 to 1918. He also quoted from Wooley,[5] who collected ninety patients, all under 21 years of age, with portal cirrhosis. The most recent report[6] of childhood cirrhosis in this country included thirty-six cases under the heading of hypertrophic cirrhosis. Many cases of cirrhosis were reported as incidental findings at autopsy, and consequently figures based on clinical evidence alone are not worth quoting.

Favoring a greater frequency of cirrhosis in childhood, Seitz[7] found 320 cases, thirty-one in infants, seventy-two among young children, and 217 among older children. Moon[8] was of the opinion that cirrhosis is much more frequent than reports in the literature would indicate, and mentioned personal communications from several pathologists, all of whom had seen, but did not report, cases in childhood. Nevertheless, the total is still relatively small. The paucity of knowledge concerning etiology and treatment makes every case worthy of mention.

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In reference to surgical procedure, we have found, in the literature available to us, only two cases where omentopexy was performed in a young child. Grinnell[9] reported a series of twenty-three cases of omentopexy for the treatment of cirrhosis of the liver. Three of these patients were under 20 years of age, the youngest being 12 years old. Hughson's series[10] of twenty-six cases included one child of 13 years upon whom omentopexy was performed. Calvin and Saffro[11] and Monerieff and Steen[12] did report operative treatment in cases of cirrhosis, but the method consisted of surgical drainage of the gall bladder. In Laurie's[13] case, a splenectomy was performed.

REPORT OF CASES

CASE 1.—R. M., an 8-year-old boy, was admitted to Morrisania City Hospital on May 23, 1935. He was referred from the outpatient department, with a history of cough since birth. One and a half months prior to admission the child complained of cervical gland enlargement which was followed by vomiting after meals, a sore throat, aggravation of the cough, night sweats, loss of appetite, constipation, and epigastric discomfort. His family history was irrelevant except for the death of an aunt from pulmonary tuberculosis. The twin of this child died at the age of six following an attack of measles and pneumonia in 1933, at which time our patient also had a severe case of measles followed by bilateral pneumonia.

Physical examination on admission revealed a pale, emaciated child who appeared to be chronically ill and cachectic. The skin was smooth and soft in texture. A few cervical, axillary, and inguinal lymph nodes were palpated. The eyes reacted sluggishly to light and accommodation. The ears, nose, and throat were normal. No murmurs or irregularities were noted in the heart. Examination of the lungs revealed some flatness at the right base and to a lesser degree at the left base posteriorly. The breath sounds were somewhat diminished over these areas, but no râles were audible. The abdomen was slightly distended. The liver was enlarged, hard, and nodular and palpable 6 cm. below the costal margin. The spleen was not enlarged. Fluid could not be demonstrated in the peritoneal cavity. The genitals were normal. No pathologic reflexes could be elicited. During the entire month the child remained in the hospital it was noted that he did not speak to any of the nurses or doctors. Questioning of the mother revealed that this lack of response was due to bashfulness. He was quite loquacious at home. The extremities showed marked clubbing of the fingers and toes (Figs. 1 and 2). Rectal examination did not disclose any abnormality.

Radiographic and fluoroscopic examination of the chest disclosed a mottled infiltration of both pulmonary bases which suggested the presence of a chronic inflammatory lesion, such as bronchiectasis. Excursion of the right diaphragm was somewhat limited. X-ray of the skull and sella turcica did not reveal any abnormality. Fluoroscopic and radiographic examination of the gastrointestinal tract was normal. There was some immobility of the duodenum due to the nodular liver. Roentgenograms of both humeri and femora were normal. The Schick, Dick, Mantoux, and Wassermann tests were all negative. The urine was normal except for the presence of a faint trace of albumin. The blood count was as follows: red blood cells 3,000,000; hemoglobin 55 per cent (Sahli); white blood cells 18,400; polymorphonuclears 50 per cent; lymphocytes 48 per cent; mononuclear cells 2 per cent. The icteric index was 6 and the Van den Bergh reaction was normal. Congo red test showed 72 per cent retention in the blood serum after

one hour. Stomach lavage for tubercle bacilli was negative. Examination of the feces did not reveal any parasites or abnormalities. Blood fat was 5.6 per cent.

On June 10, 1935, bronchoscopy was performed with injection of lipiodol into the bronchi. No evidence of foreign body was found, but considerable purulent material was aspirated from the right bronchus, which was the seat of a chronic inflammatory lesion. Two blood transfusions were administered. The patient was given parenteral liver extract which did not improve his condition. During the entire month there was no change in his physical condition. He was taken home on June 23, 1935, against our advice.

Fig. 1.—(Case 1) Photograph demonstrating hypertrophic pulmonary-osteoarthropathy of toes.

Fig. 2.—(Case 1) Showing hypertrophic pulmonary-osteoarthropathy of fingers.

On January 14, 1936, he was readmitted to our service and it was noted that, in addition to the increase in the size of the liver, he had a marked ascites and had lost considerable weight (Figs. 3 and 4). The abdomen was ovoid in shape and a definite fluid wave and shifting dullness could be elicited. The scrotum was edematous. On January 16, 3,600 c.c. of clear straw-colored fluid was removed from the abdomen by paracentesis. Cultures, guinea pig inoculations, and examination did not reveal any abnormal findings. X-ray films of the chest and long bones were normal as on previous examinations. Repeated paracenteses were performed without improvement. Rectal bleeding appeared. The child was transferred to the surgical service where transfusion was followed by an exploratory celiotomy. A

Fig. 3.—(Case 1) Photograph showing emaciation and ascites.

Fig. 4.—(Case 1) Lateral view showing marked ascites.

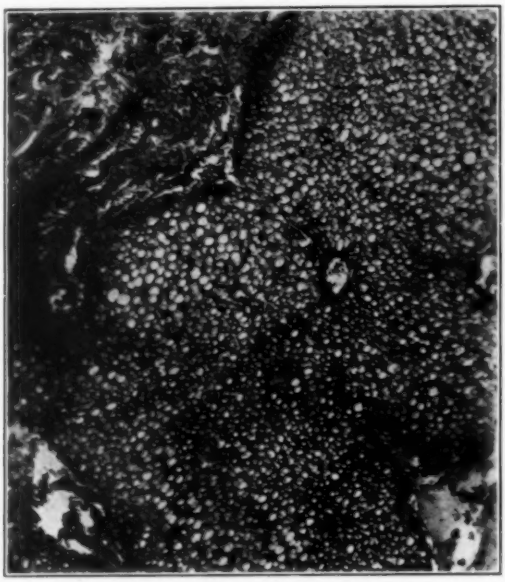

Fig. 5.—(Case 1) Section of liver tissue showing fatty infiltration, hemorrhagic extravasations near the central vein, periportal fibrosis, and regeneration of bile ducts.

biopsy specimen was taken from the liver and an omentopexy was performed. Examination of the abdominal contents was normal except for the presence of cirrhosis of the liver and ascites. The child became progressively weaker and died on March 4, 1936. Consent for autopsy was not granted.

Pathologic Changes and Sections.—The biopsy of the liver revealed a small piece of liver tissue showing large greenish areas between yellowish liver lobules. There was an occasional isolated hemorrhagic zone with grayish tissue in the midst of the hemorrhage.

Microscopic Description.—The liver parenchyma was diffusely infiltrated with fatty cells so that the liver sinuses were obliterated and normal liver cells were distinguished with difficulty. Many lobules in the region of the central vein showed extensive hemorrhagic extravasations such as usually is seen in chronic passive congestion of the liver. The periportal fields were widened due to fibrosis and round cell and polynuclear cell infiltration. There was regeneration and increase in the number of bile ducts (Fig. 5).

Diagnosis.—Fatty stage of cirrhosis of the liver.

CASE 2.—J. W., an 11-year-old boy, was admitted to the surgical service complaining of lower abdominal pain with tenderness in the right lower quadrant. The family history was irrelevant. His previous health and development were excellent. Previous illnesses included pertussis at 1 year of age, measles at 5 years, and pleurisy with bronchopneumonia at 7 years. In addition he had occasional sore throats and colds. He slept well, had a good appetite, normal bowel movements, and no digestive disturbances.

Physical examination revealed a well-nourished child 54 inches in height and weighing 58½ pounds. There was slight tenderness on deep pressure in the right lower quadrant of the abdomen. The liver and spleen were palpable. A celiotomy revealed a slightly injected appendix which was removed. The liver was found to be enlarged and studded with many wine colored nodules varying in size. One of these was excised and sent to the laboratory for examination. The spleen was found to be twice its normal size. The child made an uneventful recovery.

The physical examination on our service, after transfer from the surgical service, revealed no abnormal findings, with the exception of an irregular, palpable liver, felt 6 cm. below the costal margin. The spleen was also palpable, with a smooth edge, 8½ cm. below the costal margin. The final diagnosis, confirmed by the pathologic reports, was chronic obliterative appendicitis and portal cirrhosis of the liver.

The microscopic section of the excised nodule from the liver showed essentially normal parenchymal cells with extensive round cell infiltration, particularly in the perivascular areas. There was a moderately increased fibrosis in the periportal fields.

Laboratory Data.—The Wassermann and Kahn tests were negative. X-ray films of the chest, skull, and long bones failed to reveal any abnormality. The urine was negative.

The blood count was as follows: white blood cells 9,400; polymorphonuclears 67 per cent; lymphocytes 32 per cent; mononuclear cells 1 per cent; Congo red test showed 80 per cent retention in the blood serum after one hour; total blood proteins 6.1 mg. per cent; albumin 4.9 mg. per cent; cholesterol 187.5 mg. per cent; urea nitrogen 19 mg. per cent; sugar 95 mg. per cent.

The patient returned to us on Oct. 5, 1938, for re-examination. He had a fairly good appetite. There were no symptoms of indigestion. His bowel movements were regular. He was active and did fairly well in school. During the interval period he had had no illnesses. He had gained weight normally during this time. His only complaint was that of rather frequent headaches coming in the morning and lasting all day.

Physical examination revealed a well-developed, rather thin boy. There was no icteric tint noted, although he had a sallow complexion. There was a moderate postnasal discharge; turbinates were normal. There was slight hypertrophy of the tonsils. The heart and lungs appeared normal. The liver and spleen were definitely no larger nor firmer than on previous examinations, after an interval of eighteen months (Fig. 6). However, the liver was tender on pressure. There was a well-healed scar of the previous appendectomy. The testicles were not completely descended. His height was 57 inches and his weight 67 pounds. X-ray examinations revealed no abnormalities in the bones; no evidence of esophageal varices; but there was some cloudiness in the right antrum. Urinalysis was normal. Urobilinogen was present in a dilution of 1:4. The blood count was as follows: red blood cells 4,790,000; hemo-

Fig. 6.—(Case 2) Photograph showing the extent of splenomegaly and hepatomegaly on December 20, 1938.

globin 80 per cent (Sahli); white blood cells 8,650; mature polymorphonuclears 46 per cent; stab forms 2 per cent; lymphocytes 37 per cent; eosinophiles 15 per cent. The icteric index was 8. The galactose tolerance test revealed an excretion of 2.25 gm. in five hours. The hippuric acid test revealed an excretion of 2.7 gm. The platelet count was 350,000. There were 7 reticulocytes for every 100 red blood cells counted. The total proteins were 6.46 mg. per cent. The albumin was 4.23 mg. per cent and the globulin 2.23 mg. per cent. The albumin globulin ratio was 1.9. The serum cholesterol was 178 mg. per cent and the cholesterol ester 110 mg. per cent. The bromsulphalein test was normal.

CASE 3.—I. H., a white girl, 6 years of age, was admitted to the Morrisania City Hospital on April 23, 1935, with a history of abdominal pain and vomiting. Her previous history included measles at 2 years of age, whooping cough at 4, mumps at 5, chicken pox at 6, and frequent sore throats and colds. A tonsillectomy was performed at 3 years of age. Her appetite had always been poor and her bowels were moderately constipated. Her present illness began six hours before admission. Periumbilical pain was the first manifestation and was followed one hour later by vomiting. The pain then shifted to the right lower quadrant. She was given citrate of magnesia which she vomited. A small soap-suds enema gave no relief.

Fig. 7.—(Case 2) Infrared film disclosing no increased anastomosis of the superficial abdominal veins.

Physical examination revealed a thin, poorly nourished child weighing 35 pounds, who appeared acutely ill. Physical examination was negative except for abdominal signs. There was moderate distention with tenderness in the right lower quadrant and some rebound tenderness also. At McBurney's point a vague mass was palpated. On rectal examination tenderness on the right side was elicited. The liver and spleen were not palpable. The temperature was 99.2° F. The urine was negative. The blood count was as follows: white blood cells 17,240; polymorphonuclears 84 per cent; lymphocytes 16 per cent; hemoglobin 80 per cent (Sahli).

At operation an acutely inflamed appendix was found. The liver and spleen were normal. The patient made an uneventful recovery and was discharged on May 4, 1935.

On Jan. 26, 1936, she returned to the surgical clinic for a follow-up examination. Her condition was improved, the wound was well healed, and there were no gastrointestinal symptoms. The liver and spleen were not palpable.

On May 14, 1938, she was admitted to another institution with a history of two weeks duration of jaundice, clay-colored stools, enlarged abdomen, and epistaxis. Her condition became progressively worse and she died in cholemia on May 30, 1938. The final pathologic diagnosis was bronchopneumonia, subacute yellow atrophy of the liver with extensive nodular regeneration, and portal cirrhosis.

COMMENT

The greatest obstacle to a rational discussion of cirrhosis of the liver is the lack of standard nomenclature. Authors discussing this subject must still resort to a descriptive classification, due largely to the lack of knowledge concerning etiologic factors. This explains why such unrelated types as portal, toxic, syphilitic, and other varieties of cirrhosis are included in one group. It is obvious that some of these terms are etiological, whereas others are pathologic. The definition of cirrhosis given by Hart and Lisa[14] simplifies this problem by eliminating descriptive nomenclature. They define cirrhosis as a condition characterized by a change in the architecture of the liver lobule. They believe the disease is not an entity, but rather a secondary nonspecific reaction to various noxious agents. The factor of infection, it is generally agreed upon, plays some role in the causation of cirrhosis. The factor of hepatotoxins present in chronic alcoholism is certainly less important in children than in adults. The specific causes of cirrhosis, such as syphilis and malaria, can be ascertained without much difficulty.

The earliest pathological manifestation of liver change is rarely found. Mallory[2] claimed that certain reticular changes in the cytoplasm of parenchymal cells was diagnostic, but no one else has reported cases early enough to confirm his observations. Usually the diagnosis is not made, nor are cases seen, until nodules have already developed.

The action of various chemicals, drugs, low-grade infection, and other noxious agents, results in central necrosis of the liver lobule. The presence or absence of jaundice is dependent upon the severity of the process. Central necrosis is the first step in the development of cirrhosis. The destruction of parenchymal cells causes collapse of the liver lobule and pressure on the central vein. It is this venous obstruction which ultimately is responsible for back pressure along the portal venous system. Venous stasis leads to ascites and hemorrhoids, as well as esophageal varices. The replacement of cells of the lobule is from the periphery inward, and the inward pressure of the rapidly growing cells increases both the distortion of the lobule and the pressure in the central vein and bile capillaries.

The leucocytic infiltration is part of the inflammatory response to the irritating presence of necrotic liver cells. At this stage there is little or no increase of connective tissue. This is the pathologic picture of

toxic cirrhosis. The biopsy specimen obtained from the liver of J. W. (Case 2) showed essentially findings which place it in the category of toxic cirrhosis. The fibrosis represents the healing of necrotic liver parenchyma, and the areas of normal parenchyma indicate successful regeneration of normal architecture. Other areas demonstrate that the lobules have developed more irregularly. The extensive round cell infiltration is indicative of the inflammatory process. The gross nodular feel of the liver is due to areas of rapidly regenerating parenchymal cells replacing the destroyed cells and growing in a most irregular fashion.

The cirrhotic process, as we have seen, may become quiescent and remain in this state, unchanged, for some time. Progress of the disease depends upon the introduction of new, or the persistence of old, noxious agents. In the case of I. H. (Case 3) the development of a fatal yellow atrophy of the liver was undoubtedly due to a reinfection of an already damaged organ. Unfortunately, we are unable to demonstrate the pathologic findings in I. H., but there was a greater amount of necrosis of the lobules despite considerable regeneration. In addition, as the portal circulation becomes more and more occluded, the hepatic artery must supply blood for a greater number of cells. The loss of the more nutritive portal blood results in a diminution of various liver functions, among them the detoxifying power. A combination of poor blood supply, with attendant anoxemia and increased accumulation of metabolites, leads to fatty infiltration and degeneration. The biopsy section from R. M. (Case 1) demonstrates the advanced nature of the cirrhotic process. There is practically no normal parenchyma visible, due to the extensive fatty changes. The inflammatory response is the same as in toxic cirrhosis, but there is more fibrosis (Fig. 5).

The so-called change from the hypertrophic to the atrophic stage is more apparent than real. This shrinkage is due to proportionately greater destruction of liver parenchyma than cellular regeneration and connective tissue proliferation. The squeezing effect of connective tissue changing into scar remains a questionable process. Atrophic cirrhotic livers are often larger than the normal sized organ.

The spleen is frequently found to be enlarged with cirrhosis of the liver. Mallory[2] states that this is due to impaired venous return through the portal system. There is a stretching of the stroma and dilatation of vessels. As a result, a fibroblastic proliferation and increase of collagenous fibers leads to sclerosis of the spleen. This is to be differentiated from Banti's disease where the spleen enlarges first, and leucopenia and anemia are present. However, Eppinger[15] stated that the liver and spleen form a unitary system, and in some instances of cirrhosis of the liver the spleen may enlarge first. According to this point of view, Banti's disease ceases to be a separate entity and falls into the same nonspecific category as cirrhosis of the liver.

It is difficult to determine in the case of J. W. (Case 2) which factor might be responsible for the cirrhosis, the bronchopneumonia as an acute illness, or the chronic infection of the appendix. It is conceivable that infection from the appendix might be conveyed to the liver by way of the portal circulation. It is significant that in Cases 2 and 3 there was evidence of appendiceal inflammation. In J. W. the process was a chronic one; in I. H. it was acute. R. M. showed a much more advanced stage of cirrhosis. The presence of a chronic bronchiectasis presumably was the source of persistent infection which subjected an already damaged liver to further trauma.

One manifestation in Case 1, which has been noted in several other instances of cirrhosis of the liver, is hypertrophic osteoarthropathy. In our case the bronchiectasis may very well have been responsible for the condition. Nevertheless, Calvin and Saffro's case[11] showed this change and Mercora[16] described it in an adult with cirrhosis and jaundice. In neither case was any other chronic infection present. Marie[17] stated that the process is a symmetrical hyperostosis due to ossifying periostitis and may develop as a result of any chronic toxemia such as bronchiectasis, lung abscess, pyelonephrosis, and even hyperthyroidism with hepatomegaly (Figs. 1 and 2).

The presence of persistent gastrointestinal symptoms, such as anorexia, nausea, or jaundice, should suggest among other diagnoses cirrhosis of the liver as a possibility. It is noteworthy that in Case 2, with the exception of right lower quadrant pains, there were no gastrointestinal symptoms. Mallory[2] pointed out that passive congestion resulting from portal obstruction led to catarrhal inflammation of the gastrointestinal tract, and was responsible for the symptoms of anorexia and nausea. In Case 2, with presumable passive congestion, as shown by enlargement of the spleen, no symptoms have developed. There is no doubt that the cirrhotic process is a progressive one and has not been altered by removal of a chronically inflamed appendix. Consequently, it would seem that maintenance of normal liver function is of greater importance in the prevention of gastrointestinal symptoms than adequacy of the portal circulation.

Although leucocytosis has been frequently reported in cirrhosis of the liver, eosinophilia has never been noted as a characteristic finding in any of the other cases. In Case 2, we found 15 per cent eosinophiles in the absence of parasitic infestation or allergic phenomena. We can offer no explanation for this finding.

Jaundice as a symptom is quite significant. It was never observed in Case 1 or 2. Toxic hepatitis with jaundice later in life may be mistakenly diagnosed as simple catarrhal jaundice. Should toxic jaundice be superimposed on an already damaged liver, the end result becomes serious. This was illustrated by Case 3. No abnormality was noted in the liver or spleen while the child was under our observation, nor was

any gross pathology observed at operation. The child apparently was in good health and well compensated from the time of discharge until the toxic hepatitis developed.

As portal obstruction becomes greater, various anastomoses increase in size and number, especially in the lower end of the esophagus, the rectum, and superficial veins of the abdomen. In the esophagus large varices may result and are a potential source of profuse bleeding. Such hemorrhage was absent in all three cases. Infrared photography[18] is of value, especially in cases such as J. W., to demonstrate the earliest manifestations of portal obstruction. Where ordinary photographs may reveal nothing abnormal, infrared films plainly show an increased anastomosis of superficial abdominal veins (Fig. 7).

In regard to treatment, medical methods have not been shown to be of any great value. The consensus of opinion favoring infection as an etiologic factor suggests the advisability of eliminating chronic foci from the body. Grayzel and Radwin[19] were successful in reducing the increase in the size of the liver found in diabetes mellitus by the use of pancreatic extract (lipocaic) in cases where insulin had failed. This hepatomegaly was attributed to fatty infiltration. Administration of this pancreatic extract might possibly be of benefit in cirrhosis of the liver where fatty infiltration is one of the pathologic processes. Rosenberg[20] reported a case of fatty metamorphosis of the liver with marked hepatic insufficiency which was improved by lipocaic. High carbohydrate diets are of questionable value. Liver extract administered parenterally proved to be of no value in Case 1.

By improving and increasing the anastomosis between portal and systemic venous systems through omentopexy the danger of hemorrhage from overloaded varices may be lessened. In Grinnell's[9] group of twenty-three cases of omentopexy which included mostly adults, the results were not encouraging. Hughson[10] was also pessimistic about the value of surgery in cirrhosis. To be of benefit, it is important that the operation be done early in the disease. Case 1 was operated upon apparently too late, for by that time liver function was inadequate. Nordland and Larson's patient[21] obtained successful palliation for two years and then died of an intercurrent infection. Rhoads and Stein[22] reported a case of Banti's disease where splenectomy and ligation of the coronary artery and vein resulted in diminution in size of large esophageal varices. Calvin and Saffro[11] and Monerieff and Steen[12] found excellent improvement following cholecystostomy.

The prognosis in cases of cirrhosis is dependent upon maintenance of liver function and the extent of anastomosis between the portal and systemic systems. The point of greatest danger is in the vicinity of esophageal varices, which may rupture and cause fatal hemorrhage. Intercurrent infection very often is responsible for the death of a child

with cirrhosis. The debilitated state, wherein anemia, poor nutrition, and venous stasis are found, provides scant resistance to such infections as bronchopneumonia.

SUMMARY

Three cases of cirrhosis of the liver have been presented, with pathologic studies. The pathogenesis of portal cirrhosis has been discussed in relation to the various stages of the disease. One case represents the condition known as toxic cirrhosis, which is the first stage in the cirrhotic process. The second case demonstrates the gradual progression to an end stage in the presence of a chronic infection of the lungs. The third case illustrates rapid progression of the cirrhosis due to a superimposed acute infection of the liver.

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NARCOLEPSY

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NARCOLEPSY is a functional disturbance which is being reported more frequently every year. It is still practically an unheard of diagnosis in childhood.

A narcoleptic patient is affected by diurnal naps occurring frequently and persistently. Probably the first good description of this disease complex was given by Westphal in 1877. He was reluctant to dismiss the peculiar attacks with the expression "epileptoid."

Gileneau, in 1888, published a report recognizing this condition as a distinct entity and proposed the name "narcolepsy." This term was used, however, very indiscriminately until in 1907, when Kamp described the complete narcoleptic syndrome. Since then contributions by observers from all over the world have made familiar the various symptoms of narcolepsy. The more recent studies have been by Janota, 1930, and Daniels, of the Mayo Foundation, in 1934. The latter, in his thesis on this subject, gives a very complete description and discussion of the condition.

In reviewing the literature, a great variety of symptoms were noted, in fact, so great a variety that the identity of the condition was at times lost. The person with after-dinner drowsiness and the person who falls asleep while driving an automobile may be included, according to some. The sleepiness has nothing to do with rest obtained at night.

The case here reported is believed to be one of true and idiopathic narcolepsy without catalepsy. It is unusual because it occurs in a young girl.

The patient is a white female, born Dec. 12, 1925. She complained of repeated diurnal naps coming on regardless of the amount of sleep at nights. Because of this complaint she entered the Out-Patient Department of the St. Louis University Group of Hospitals at the age of 7½ years.

Present Illness.—In November, 1932, two months after the child had hypertrophic tonsils and adenoids removed, her mother noticed that she was gaining weight too rapidly. In January, 1933, the gain in weight was most pronounced, and attacks of drowsiness were very noticeable. The mother took the child to the doctor, who prescribed a tonic. The condition became worse; the child began to do poorly in school, moving from first place to almost last in a period of three months. The teacher complained to the mother that the child paid no attention in class and was continually napping.

The mother noticed at the same time that the child had ceased riding her bicycle and using her roller skates. Upon returning home from school she preferred to sleep or to sit and read. In June, 1933, the child fell asleep in the bath tub on more than one occasion, and the mother had begun to fear to leave her alone. At this time she came under observation at the St. Louis University Out-Patient Clinic. She had lost

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her ability to talk properly. Her words were slurred and mumbled, and she could be understood with difficulty. This was especially pronounced when the child became excited. However, she was not nervous; she slept well at night. Her gastrointestinal tract and genitourinary tract were apparently functioning normally.

Past History.—The child has had no contagious or infectious diseases; she has had no serious illness. Her tonsils and adenoids were removed in 1932 because of an occasional earache. Her ears, however, had never discharged. There was no history of birth injury or other trauma.

Family History.—Mother and father were separated. The child lives with mother and 16-year-old sister. The mother is small in stature but not obese.

Physical examination revealed a chubby girl aged 7 years with a noticeable abdominal protuberance and strikingly robust appearance. She was 46 inches tall and weighed 55 pounds. Her *head* showed no abnormalities of size or shape. The *eyes* reacted to light and accommodation; there was no conjunctivitis, nystagmus, or strabismus. *Nose* was not infected—no discharge; *teeth* were in good condition; *tongue* was graphic. *Tonsillar fossae* were clean. No glands were palpable in the *neck*. *Facial muscles* functioned equally well on both sides and in harmony. *Pectoral* subcutaneous fat was moderately prominent for the child's age; minimum palpable rachitic rosary was found. *Heart rate* was regular, no murmurs. The heart lay within midmammillary line to the left and was not demonstrable on percussion to the right of sternum. On percussion the *lungs* were normally resonant, with no râles, and breath sounds, normal. The *abdomen* showed striking girdle fat and had a circumference of about 34 inches; palpation revealed no tender glands. *Genitals* were normal, with no pubic hair. The *arms* and *legs* were straight, with no deformity or limitation of motion. The *wrists* were obese, and fingers strikingly pointed. No eruptions or sores were seen on the skin.

Neuromuscular examination showed that knee jerks, biceps and triceps reflexes, and Achilles reflex were all within normal range of activity. Her *facial expression* was intelligent and cheerful, but her *speech* was strikingly slurred and inarticulate. The child did not seem to have complete control of the use of the tongue in speaking and was conscious of the speech difficulty. At this time the impression was that the child was suffering from a thyropituitary disturbance. Dr. Sexton of the Medical Department of the St. Louis University was called in consultation.

Laboratory Examination.—Roentgenograms of the skull revealed no pathology. Sella turcica was of normal size. Blood calcium was 9.8 mg. and blood sugar 90 mg. per 100 c.c. of blood. Roentgenograms of the elbows and the wrists revealed no retardation of the appearance of the centers of ossification. White blood count was 8,000; red blood count, 4,600,000.

Since child had no physical ailment but had the physical development of hypopituitarism, namely, marked obesity and pointed fingers, it was decided in consultation with Dr. Daniel Sexton, endocrinologist, that pituitary extract, both anterior and posterior, was indicated and, because of the extreme fatigue, thyroid extract might be tried. Treatment was begun with 1 c.c. of antuitrin and 5 minims of obstetric pituitrin given hypodermically twice a week and ½ grain of thyroid by mouth daily.

Progress.—On Aug. 1, 1933, the child weighed 58 pounds and on August 14, 57 pounds. Her pulse was regular, and temperature normal. Thyroid medication was increased to ½ grain twice daily, and pituitary injections were continued. For progressive weight changes, see Chart 1. Child's weight, recorded every two weeks, was definitely retarded in its increase during the ensuing five months, but the girdle obesity remained. The child could not tolerate a further increase in dosage of the thyroid extract and reacted by developing palpitation, rapid heart rate, nervous emotional instability, and a tremor in speech. In February, 1934, the child began to develop a severe reaction following the injections of obstetric pituitrin, which had been increased to 1 c.c. The reaction was characterized by severe headaches, severe

stomach-aches, and collapse within fifteen minutes after injections. Under this regime the periodic drowsiness was not improved. The child did not progress satisfactorily in school. However, naps did not increase in frequency, and she could easily be aroused. No cataleptic seizures occurred. The difficulty in speech remained. The *conclusion* was "no improvement."

In March, 1934, thyroid and pituitary extracts were discontinued and ephedrine sulphate, ⅛ grain t.i.d., was given. In the ensuing week child seemed slightly improved. Ephedrine was increased to ⅜ grain twice daily. The child began to play after school in preference to reading, and she seemed more alert in class. Two weeks later ephedrine sulphate, 3 grains t.i.d., was ordered. Speech became definitely improved: words were spoken more clearly. Emotional instability seemed to have disappeared. During the ensuing six months, the above treatment was continued. The child improved rapidly in school, being advanced an extra grade. She began to roller-skate and to ride her bicycle. She had only an occasional desire to nap. Ephedrine

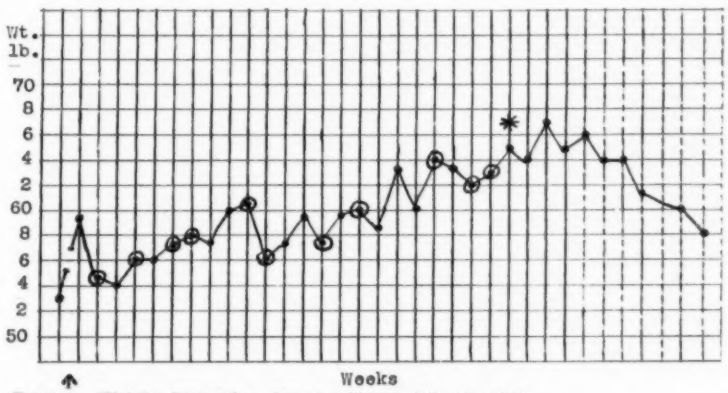

Chart 1.—Weight fluctuation, Jan. 1, 1933, to July 28, 1934.
° Administration of thyroid plus anterior pituitary and obstetric pituitrin.
* Previous medication stopped; ephedrine begun.
Arrow indicates the lapse of about 6 months.

sulphate was then reduced to ⅜ grain twice a day. Her symptoms did not reappear. Girdle obesity disappeared and child gradually changed into a well-proportioned girl a few pounds over the average weight for her height and age. In January, 1935, ephedrine sulphate was replaced by neosynephrin hydrochloride, 10 minims t.i.d. Child rapidly developed sleepiness, laziness, and the tendency to slur in her speech. During previous ten months whenever mother stopped medication for twenty-four hours, the child reverted to original syndrome. Consequently, the child had become so dependent on the use of ephedrine that she objected to any further experimentations with other products and even was reluctant to reduce the dosage if that were requested. In December, 1936, the child began again to put on weight rapidly and returned for re-examination on this account. At that time she presented the picture of a precocious child who had begun to menstruate at the age of 10 years and who was at the head of her class in the latter part of the sixth grade. In this instance, increased obesity was more related to excessive eating occurring during a natural period of rapid growth. At this time the child had not received ephedrine for about six months. Ephedrine, ⅛ grain three times a day, was again ordered because the child was complaining of fatigue and lack of desire to play. Again with the administration of the ephedrine sulphate these symptoms rapidly disappeared. One month later ephedrine was discontinued without any untoward results.

DISCUSSION

From the cases reported by Daniels and from his reported literature, narcolepsy is a disease of the young adult. He reports only five cases in patients under 10 years of age and further that the condition is very rarely seen in the female sex. Yet the case here presented is that of a girl with the onset of the condition at 7 years of age. The prognosis is apparently rather dubious. Complete cures are reported as rare and indefinite. Spinal puncture, encephalography, and various stimulants, such as caffeine and strychnine, have been used, the former with apparently curative value in isolated cases, the latter without avail. The success of a spinal puncture or encephalography would indicate that increased intercranial pressure played a role. However, this is not demonstrable. From the facts known at present, no conclusion can be reached as to the definite etiology of the condition. A great number of the cases follow encephalitis. Various authors think the disease is due to some change in the thalamus or in the region of the third ventricle. It is interesting that ephedrine sulphate should be of such distinct value in relieving symptoms when the blood pressure and basal metabolism are so varied that they apparently have no definite relationship. It is unusual that such a drug should aid the redistribution of fat as seen in this case, unless it aided in stimulating the pituitary to function properly or so increased the metabolic rate to prevent such deposits. However, there are favorable reports on the use of ephedrine. Daniels reports distinct evidence of its use, and so does Zahorsky, in his case of a child.

In this particular case an endocrine disturbance may play a role, or it may be just coincidental as it is in many cases. There is no evidence of severe infection or encephalitis in the history. Daniels reports the onset following an injury to the head.

The prognosis is evidently excellent in this particular case as observation during 1935 and 1936 has indicated. The observation over the same period of time indicates that the ephedrine sulphate given for an extensive period has in no way produced any toxic effects on the child.

SUMMARY

A typical case of narcolepsy occurring in a white female, 8 years of age, is presented. The symptoms were not influenced by thyroid or pituitary preparations but were promptly relieved by ephedrine sulphate. After the administration of the ephedrine for a period of about a year, treatment was no longer necessary, and the child was symptom free with the exception of two months recently when symptoms appeared and necessitated the use of ephedrine therapy again. However, this medication was necessary for only a period of about one month.

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DECREASED SUGAR TOLERANCE DUE TO INFECTION

An Unusual Case of Acute Appendicitis Complicated by Unexpected Discovery of Diabetic State

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K. W., aged 8 years, was first seen at 4:30 A.M., on Nov. 21, 1938. History obtained from his uncle was as follows: The boy had been perfectly well the day previously and in good spirits. He had had his usual supper and had gone to bed feeling well. He was awakened about midnight with pain in the "pit of the stomach" and "on the navel." The pain was severe enough to make him cry out and vomit. In about two hours the pain settled in the right lower quadrant. The uncle thought that the boy had had a similar attack one month ago. This was confirmed later by the boy's mother.

Personal History.—One brother, 6 years old. One sister 3½ years old.

Past Health.—No operations, no illnesses. The boy has been a well child.

Family History.—Nothing of note.

Physical Examination.—Tonsils ragged and mildly inflamed. Chest clear. Heart, genitalia, limbs and joints normal. The right side of the abdomen was tender to pressure and there was definite muscle splinting. This was most marked in the lower quadrant. Pressure on the left side referred tenderness to the right. There was definite rebound tenderness. Rectal examination was unsatisfactory. Brudzinski and Kernig signs were negative. Temperature, 100.2° F.; pulse 100; respirations 35.

Laboratory Examinations.—White blood count was 17,900. Urinary specimen examined about 5 A.M. was as follows: specific gravity, 1023; reaction, alkaline; albumin, negative; sugar, 3 per cent; acetone, 1 plus; micro, amorphous phosphates.

In view of the unexpected findings in the urine, impending diabetic coma suggested itself as a cause of the abdominal pain in the differential diagnosis. To make sure that the sugar found was not an artefact in a bottle already containing sugar (the aceto-acetic acid could be explained by the vomiting), an attempt was made to obtain a fresh specimen by giving the child water to drink. He readily vomited. About 8 A.M. he was catheterized. The specimen this time contained a 4 per cent sugar and a four-plus acetone (on a basis of xxxx). A diagnosis of acute appendicitis and diabetes was made.

While the operating room was being prepared, 500 c.c. of saline was administered intravenously and sixteen units of insulin was given subcutaneously. Ethyl chloride induction and ether anesthesia were administered. Through a low pararectus incision the abdomen was opened. Free pus poured out. About one cupful was removed. No mass, no cecum nor appendix could be palpated. The incision was enlarged and still nothing could be found. The incision was again enlarged, practically to the costal margin. By tracing the small bowel, what was taken to be the cecum was found practically at the hepatic flexure, but very close to the center of the body. The appendix was not visualized. The cecum was retracted gently to the midline. Just below the gall bladder on the posterior abdominal wall a bead of pus stood out. Pressure about this area brought another bead of pus. Extending from this area to the under surface of the gall bladder were two rather strong "string adhesions." With careful searching a wormlike body could be felt deeply retroperitoneal,

paracecal. It was completely imbedded, no mesentery whatsoever. The junction of this body with the cecum was mobilized and cut between two ties. The cecal stump was inverted. The rest of the appendix was traced to the spot where the pus came from. This was taken to be the tip. To my surprise it was not the tip, but a markedly acute kink. The remainder of the appendix about five inches long, doubling back on itself, was retrocecal. This was virtually dug out with the forefinger. The string adhesions to the gall bladder were cut between ties. The abdomen was closed in layers and one drain through a stab wound in the right flank.

The subsequent course was rather stormy for a few days postoperatively. Neo prontosil parenterally followed by sulphanilamide by mouth soon brought the infection under control. The subsequent period became one of constant improvement. Every specimen of urine was tested for sugar and aceto-acetic acid. Blood sugar estimations were done as a check. Insulin was administered, one unit for every 2 gm. of carbohydrate taken. At first the only carbohydrate given was parenterally. November 28, the patient was sugar-free and acetone-free with no insulin. Later, twenty-four-hour specimens contained no sugar nor aceto-acetic acid on an uncontrolled diet.

On Dec. 21, 1938, after he had been up and about for ten days and perfectly well, a blood sugar curve was done after administration of 30 gm. of glucose in lemon juice. The curve obtained was interpreted as an abnormal type and indicated a lessened sugar tolerance.[14] The patient's mother was told that the boy had a decreased sugar tolerance and as such, to restrict an excessive sugar and candy intake. Carbohydrates in general were not restricted. She was also told that with subsequent infections this boy would more than likely show a decreased carbohydrate tolerance.

On Jan. 14, 1939, he had 0.1 per cent sugar in his urine on an uncontrolled diet.

In a discussion of the above case it would be ideal if one had a blood sugar curve taken previous to the onset of the boy's illness. As it is, one can only go on the history given from a dependable source. Repeated questioning only served to show that there had been nothing to suggest a decreased sugar tolerance previous to the onset of the illness. Two interpretations may be taken. Either this child was perfectly normal before the onset of the illness and had his sugar tolerance decreased to the state where he can be considered a very mild diabetic, or he was a potential diabetic and has had his sugar tolerance mildly decreased.

When the boy was first seen the sugar in his urine was 3 per cent with a one-plus acetone. About three hours later when a second specimen was obtained the sugar was 4 per cent and the aceto-acetic acid four plus. Where before he had been rather bright and responsive, he was now quite dull, his respiratory rate quite rapid. He was going slowly into coma. With the drainage of the abdomen, parenteral fluids with insulin administration, the sugar tolerance rapidly improved. Immediately postoperative his blood sugar was 0.267 per cent. A preoperative estimation was not done. That night it was 0.113 per cent. Incidentally he was typed and a donor obtained should the necessity for transfusion arise. On the evening of November 22, thirty hours postoperative, he had a severe insulin reaction going on to a convulsive state necessitating 25 c.c. of 25 per cent glucose intravenously. This certainly was a rapid improvement in sugar tolerance, and showed that if the

pus had not been drained the boy would most certainly have gone into coma in spite of the concomitant treatment of the diabetic state.

One can try to explain the pathologic condition found in the abdomen; certainly an abscess containing one cup of pus did not form in ten hours. This boy had an attack of acute appendicitis lasting five days one month before, and an abscess formed about the inflammatory area. This is not altogether conjectural. The mother volunteered the information that the boy had been limping ever since his previous attack of pain in the abdomen. The abscess was resting on and irritating his right psoas muscle. At midnight of November 21, for some reason or other, the already obstructed appendix developed increased peristalsis and ruptured releasing the pus into the peritoneal cavity with resultant pain in the right lower quadrant. Stephenson[17] reported a case of retroperitoneal appendicitis with abscess formation posteriorly and a fatal outcome. It was fortunate in our case that the abscess pointed forward and ruptured into the peritoneal cavity so that it could be drained.

In diabetics, pain in the abdomen brings up differential diagnosis of three distinct entities: insulin shock, coma, and acute surgical condition of the abdomen. Here, of course, one could rule out insulin shock because the boy had not taken any insulin. The remaining two signs and symptoms because of their resemblance can be confusing. White[20] states, "An acute surgical abdomen provides . . . pitfall in differential diagnosis." In both conditions there are pain, nausea, vomiting, fever, leucocytosis, and if there is much vomiting the abdominal muscles may become tender with resultant splinting. The white blood count drops to normal in twenty-four hours in coma when it responds to treatment. However, if a surgical abdomen is really feared, it might be dangerous to wait that long. Under the circumstances White[20] suggests that the abdomen be opened under local anesthesia. Two patients were operated on while they were in coma, with no harmful sequelae.

John[9] gives the following description of the onset of diabetic coma. "The first complaint is that they get sick to their stomach and vomit. Abdominal tenderness can frequently be elicited before they drift into unconsciousness. Is this tenderness a pancreatic tenderness due to the affection of the pancreas, or is it just a plain abdominal tenderness? Both views will be found expressed in the literature. In a non-diabetic individual after a siege of vomiting, abdominal tenderness can be elicited, for the muscle spasms produced by vomiting are enough to produce 'soreness' in the abdominal area." He reports two patients with acute abdominal pain. One was erroneously operated on for acute appendicitis, and a normal appendix removed. The child died in coma the next day. Another child missed being operated on for acute appendicitis. It died the next day in coma. There was no post mortem to

prove that this was not an acute abdomen. John[9] in his differential diagnosis of diabetic coma does not mention acute appendicitis.

It was of the utmost importance in our case to have a correct diagnosis. One felt that in spite of the marked glycosuria and acetoacetic acid in the urine, an acute infection was present in the boy's abdomen. To arrive at this decision one had only two factors to go on: (1) a recurrent typical history of acute appendicitis, a generalized abdominal pain which in a few hours settled out in the right lower quadrant; (2) the finding of absolute localized tenderness. All other findings which one considers in a case of acute abdominal pain could here be caused by an oncoming diabetic coma. As such they could be misleading. I have recently operated on two patients with cases of proven acute appendicitis which were rather instructive. They brought out the point that of all the signs and symptoms in the diagnosis of an acute appendicitis, localized tenderness stands supreme. One was in a girl aged 16 years. She was seen with a history of acute onset of generalized abdominal pain. This soon settled out in the right lower quadrant. The temperature, pulse, and white blood count were normal. *There were definite tenderness and rigidity.* A similar attack had passed off five days before. Medical attention was not sought the first time. In spite of the lack of signs of acute infection she was operated on that afternoon. The appendix was about three and one-half inches long. The proximal one inch was perfectly normal. The distal two and one-half inches were dilated to about double the diameter of the proximal part and full of thick heavy pus. No obvious cause was found for the change in caliber of the distal segment of the lumen. There was no obstruction anywhere in the lumen of the appendix. Light pressure on the bulbous part moved the pus to the proximal part and while it was still attached to the cecum could easily move there. In other words the pus pocket was well drained and hence no absorption. A few days later a child 2 years of age was seen in consultation. The history was that about twenty-four hours previously the child began to have frequent bowel movements and began to vomit. About sixteen hours later pain developed in the abdomen. On examination eight hours later the temperature was 103° F., and pulse, 120. The only important finding was that the child was irritable and did not want to be touched. The whole abdomen was held rigid. With careful handling the abdomen softened all over except in the right lower quadrant. It remained splinted, with marked tenderness. The white blood count was 24,000. At operation a markedly inflamed appendix with adherent omentum surrounded by a pocket of pus was found.

Both of the foregoing cases had acutely inflamed appendices, yet the only common denominator in their diagnosis was localized tenderness. It was entirely on the basis of the finding of localized tenderness,

supplemented by a history, that our patient was operated on in the face of a dangerous situation.

REVIEW OF RECENT LITERATURE REGARDING INFECTION AS A CAUSE OF DIABETES

In children, and even in infants whether they be diabetic or normal, infection is a big factor in decreasing sugar tolerance. In normal children an acute infection can cause glycosuria and rise of blood sugar. When the system is burdened with an extra load of sugar a diabetic curve results. This decreased carbohydrate tolerance may last months. As in true diabetics the tolerance is increased by administration of insulin.[1, 4, 9, 20] Blood sugar regulation is so sensitive that rise in blood sugar has been seen before temperature rise. Blood sugar fluctuation follows temperature.[18] Suppose now that a potentially diabetic child develops an acute infection. With the subsidence of the infection, one of two things can develop. Either he comes back to the state where he was prior to the infection or his tolerance may be further reduced to actually remain in a diabetic state.[9] Low-grade and early diabetic manifestations are not infrequently first diagnosed during acute infections.[11] Again a mild diabetic may have his tolerance so badly decreased that his case becomes a severe one or even be precipitated into coma. What happens to a reasonably severe diabetic during an infection is only too common knowledge.[9]

White,[20] in her series, found surprisingly few acute infections in the year prior to the onset of the diabetic state. However, in a series of 100 children, only 2 per cent were free of any sort of infection at any time preceding the onset of the diabetes. She suggests that the effects of the infection found, as tonsillitis, measles, pertussis, mumps, pneumonia, scarlet fever, chicken pox, jaundice and appendicitis, may have been latent and the results appeared later. In her experience[19] diabetes is rare in private school children. Because of better financial background their foci of infection would be taken care of better and sooner.

The type of infection accused is varied. Acute infections are most commonly indicated. Geyelin[6] stated that he had seen eight cases in which diabetes arose within five weeks after acute infection. Joslin found a history of antecedent infection in about 15 per cent of his cases (quoted by Williams and Dick).[21] Newcomb[13] in his series of cases found diabetes followed such infections as otitis media, measles, peritonsillar abscess, and varicella. The average length of time between the infection and the onset of diabetes was about one and one-half months. Lierle and Potter[11] incriminate chronic and subacute infections of the upper respiratory tract as a cause of diabetes in children. Chronic and subacute infections are more readily diagnosed in children by thorough otolaryngologic examinations and with their eradication the carbohydrate tolerance improves. A diabetic child should receive the same careful search for foci of infection as a nephritic or arthritic patient.

In any one particular case of diabetes, the role infection plays can only be judged on circumstantial evidence. Only in the exceptional case is it possible to give proved evidence that before a certain date the child was free of diabetes and following this illness diabetes developed. Here and there one is able to pick out such an occasional case. Medovy[12] reports two cases; one developed diabetes during mumps and another just after an attack of chicken pox. Blaisdell[2] reports an interesting case of a child who developed poliomyelitis early in October. She was discharged seven days later entirely well. Examination of the urine was negative on both admission and discharge. During the middle of November of the same year she was admitted to hospital in coma. She recovered and remained diabetic, necessitating the use of twenty-seven units of insulin daily. Rennie[16] reports a child with mumps. Convalescence was incomplete and within three months the child had pronounced symptoms of diabetes.

Influenza,[9] acute colds,[9] tonsillitis,[1, 9, 13] epidemic parotitis,[9] typhoid,[1] varicella,[13] otitis media,[13] scarlet fever,[3, 21] cholera,[17] diphtheria,[21] malaria,[21] rheumatic fever,[12] are a list of diseases following which cases of diabetes mellitus have developed. Some of these deserve special mention. Following an epidemic of influenza, the incidence of diabetes has been observed to have increased.[9] Barach[1] in an analysis of a series of cases concludes that chronic tonsillar disease and typhoid fever play an important etiologic role in the development of diabetes mellitus. Boyd (quoted by Penwell[15]), feels that diseased tonsils are a very important factor. Gundersen[8] found that in Norway following mumps epidemics there was a definite increase of diabetes mellitus in the age groups suffering most from mumps. This occurred following several epidemics. He suggests the virus that causes epidemic parotitis also attacks the pancreas. Atrophy of the pancreas follows the inflammation similar to the atrophy of the testicles. Ettinger and Reed,[5] working on the premise that there may be a filterable virus in the urine of diabetic patients, injected Berkefeld-filtered urine into rabbits and dogs under various conditions. There were no changes in the blood sugar nor was there any glycosuria. They conclude that Berkefeld-filtered urine of a diabetic patient contains no organisms which can reproduce the diabetic condition in dogs or rabbits. As a contrast to this, Bergey (quoted by Ettinger and Reed[5]) two years previously in 1926, was able to produce glycosuria in rabbits by injecting in them Berkefeld-filtered urine from diabetic patients. No blood sugar estimations were made. Gilchrest and Wilson[7] incriminate the staphylococcus and state, "We are now convinced that in most diabetics in this region we are dealing with a staphylococcic factor and that by using (staphylococcus) toxoid we are attacking the cause."

The above review of infection as a cause of diabetes mellitus is brief and gives just the salient points. What is the explanation of hyperglycemia in infection, whether it be in the normal child, the potential diabetic, or the diabetic? Five possibilities present themselves.

1. Reduced insulin output.
2. The action of the insulin may be interfered with as it is in acidosis and fever.
3. Inability of the liver and muscles to store glycogen readily.
4. The nervous control mechanism may not be functioning properly.
5. There may be an increased output of adrenalin.

A reduced insulin output is the generally accepted explanation of hyperglycemia in infection.[9, 21] Exogenous insulin improves the carbohydrate tolerance. Liver damage is not likely to be a factor because in patients with marked cirrhosis of the liver, there is no hyperglycemia. That one factor only is at fault to the exclusion of the others is not entirely correct. All five are probably involved to different degrees in individual cases.[9, 21]

Most authors give infection a small place as a factor in the etiology of diabetes.[21] That infection can cause decreased carbohydrate tolerance, none can deny. Briefs of cases and statistics have been presented. These more than suggest the part which acute and chronic infections play in the actual production of diabetes mellitus. Rabinowitch (quoted by Williams and Dick[21]) in a statistical study found, "Nine times as many patients with gall-bladder disease had diabetes as had patients in general." Stanfield and Warren (quoted by the same source) found at post mortem of two diabetic children, an infiltration of the islets of Langerhans. These findings along with the histories of the two cases suggested an infectious origin.

SUMMARY

An unusual case of appendicitis and recognition of a diabetic state for the first time is presented. The diagnosis and treatment are discussed and facts and findings correlated in retrospect. The recent literature regarding infection as a cause of diabetes mellitus is reviewed.

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AURICULAR FLUTTER IN A YOUNG INFANT WITH RECOVERY ON DIGITALIS THERAPY

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THE importance of the invention of the electrocardiograph by Einthoven can hardly be overestimated. No one will deny that with this apparatus valuable data of the cardiac function are obtainable. Yet it is a fact that the electrocardiograph is little used in pediatric practice, particularly for the study of heart disease in infants. That in some infant clinics an electrocardiograph is as yet to be found speaks volumes in this respect.

Most of the cases of heart disease in infants are congenital cardiac defects, and it is a well-known fact that the electrocardiograph is not of value in the diagnosis of valvular trouble. It is possible in these cases, to be sure, to demonstrate a particular ventricular preponderance, the knowledge of which is not very important. The existence of dextrocardia in infants can be corroborated with certainty, although the significance of this diagnosis for the affected individual is about nil. In rare instances, as a curiosity, congenital heart block is found in a newborn infant, a finding which might indicate definite effective therapy only in the case of a syphilitic lesion of the bundle of His.

In the case of acquired heart disease in infants, the use of electrocardiography is of no greater importance, though of late years with it one has been able to judge more or less accurately the condition of the heart muscle. Here, too, nothing further is obtained than diagnosing the preponderance of one of the halves of the heart, while the arrhythmias, the bradycardias, the tachycardias, and the disorders of the conduction system (because of the absence of arteriosclerosis) can only be seen very rarely or not at all. Instances of these anomalies in infants have been described now and then, though these were mostly of scientific, rather than of practical, significance.

Therefore it seems important to us to record the following case of heart disease in an infant, which not only was diagnosed by means of the cardiograph, but in which the apparatus also gave the indication for the application of the therapy that led to a successful outcome.

CASE REPORT

R. S., 2 years of age, was admitted Jan. 5, 1936, with pneumonia. On admission she was dyspneic and cyanotic and had a high temperature and a very rapid pulse. The liver reached to the navel, and the spleen was moderately enlarged.

She was treated in the usual way. An injection of 0.5 c.c. of digalen was administered, and on the subsequent days 4 drops of digalen by mouth. With this medication she made a fairly quick recovery; the congestion of the lungs decreased, and

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on January 14 the frequency of the pulse was 90 a minute. Digitalis medication was then stopped. On January 18 the pulse accelerated again without a temperature rise or development of other symptoms which could account for this. On January 25 the heart rate was 165. On closer examination the heart appeared to be enlarged; no cardiac murmurs were audible. The congestion of the liver had increased again (Fig. 1). Digalen was again administered, and the pulse receded to 100-120. On February 3 the pulse appeared irregular. After every third beat there was one lacking. A remarkable observation was that during the first contraction of every series a systolic murmur was audible, which was not heard with the following beats.

Fig. 1.—R. S. before digitalis therapy. She is just over 2 years old (April, 1936). Enlargement of liver and spleen.

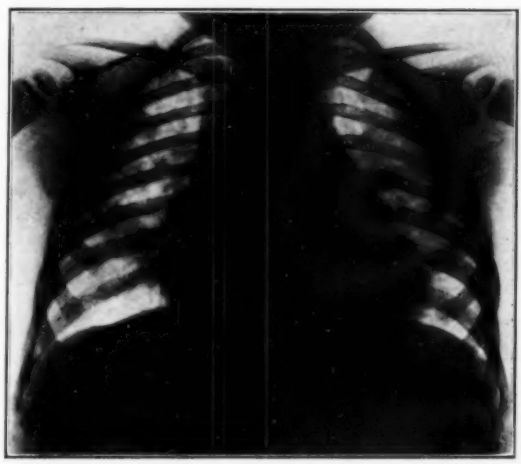

Fig. 2.—X-ray film taken during period of flutter (February, 1936); enlarged heart.

After a fortnight the pulse dropped to 62, the administration of digitalis was stopped, and metrazol was given. Then the pulse rose again to 160-170. An x-ray film of the thorax at this time showed the heart considerably enlarged both to the right and to the left (Fig. 2).

The first electrocardiogram was made in April, 1936, and it showed auricular flutter with 2:1 block, as is manifest in Leads II and III (Fig. 3.4). Attempts to influence this tachycardia with quinidine were unsuccessful.

The term *flutter* is used in the case of a rapid, regular, and coordinated action of the auricles, manifesting itself in the existence of regular pronounced auricular complexes (P-waves) in the electrocardiogram. Since the ventricles are rarely able to respond to each auricular impulse, two or more are followed by one ventricular one. Thus in the electrocardiogram a series of P-waves are seen, every second, third, or fourth of which is followed by a QRS complex.

In *fibrillation* there is an irregular undulatory movement of the contraction wave over the auricles, which then are no longer brought to contraction. However, in cases of fibrillation, fairly regular P-waves are often seen in the electrocardiogram for a while, which again are followed by a period without regularity. The term fibrilloflutter is used in these instances.

In the subsequent two years it was evident in the electrocardiograms that flutter was still present. At home the child passed through all sorts of illnesses: otitis, measles, pneumonia, all of which she sustained very well. From time to time there were attacks of dyspnea. We often saw her playing in the street where she lived, and we were always struck with the slight dyspnea and cyanosis. In November, 1938, we saw her back in the same condition. We resolved once more to make an attempt to stop the auricular flutter with the help of digitalis.

In treating auricular flutter with digitalis one employs the drug's property of slowing the conduction in the bundle of His. By this, fewer auricular impulses are transmitted to the ventricle with the result that the pulse rate decreases. One might feel satisfied with this and let the patient keep a rather regular normal pulse rate with the continual help of a small dose of digitalis. But it is possible to obtain a still better result. If the patient does not begin to show signs of digitalis intoxication, the administration of a large dose of digitalis may be continued. The flutter rhythm is then seen to pass suddenly into fibrillation. As soon as fibrillation, which must be controlled electrocardiographically, manifests itself, the medication of digitalis should be stopped. In a great number of cases after several days, sometimes even after a few hours, the normal sinus rhythm returns, and the electrocardiogram does not show a single anomaly. We intended to obtain this result in our patient.

The child was given at first 75 mg. and later 150 mg. of powdered digitalis leaf. It produced a gradual decrease of the pulse rate; after ten days it had slowed from 165 to 120. The electrocardiograms, which were made at short intervals, showed progressive blocking of the auricular impulses. The relation between auricular and ventricular rates was connected with the respiration: during inspira-

tion it generally amounted to 2:1, during expiration to 4:1 (Fig. 3*B*). The dose of 150 mg. of digitalis a day was continued for some weeks. On the twenty-third day of digitalis medication, the pulse was 80 and the rhythm had suddenly become regular.

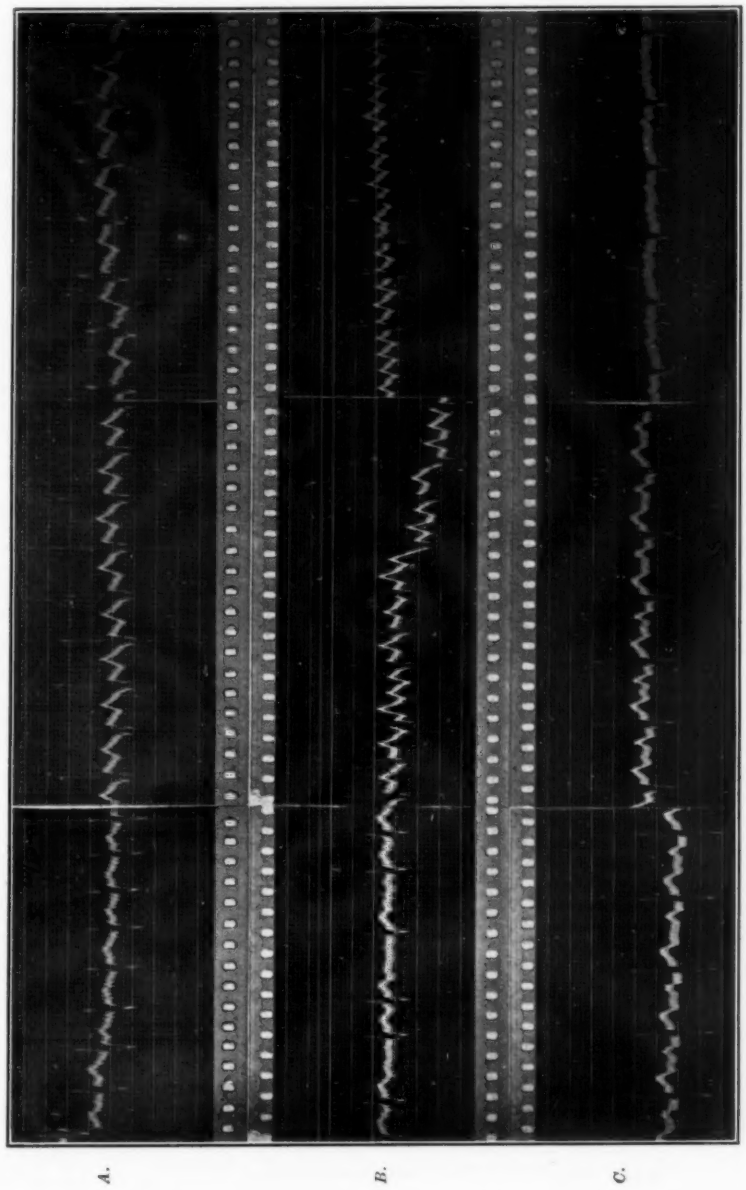

FIG. 3.—Electrocardiograms: *A*, auricular flutter with 2:1 block (April, 1936). *B*, during digitalization, with 2:1 to 4:1 block (November, 1938). *C*, normal rhythm restored (December, 1938).

An electrocardiogram made then showed a normal sinus rhythm. The P-Q distance was slightly lengthened, which is regarded as the effect of digitalis (Fig. 3C). No further digitalis was administered and for another five days 150 mg. of quinidine was given. The sinus rhythm continued with a frequency of 90. Signs of cardiac failure were hardly found any more: breathing had slowed to the normal tempo, and while the liver had shrunk considerably, it seemed, nevertheless, to be still larger than normal; the spleen was no longer palpable.

Auscultation revealed a distinct systolic murmur, the maximum intensity of which was situated in the third intercostal space to left of the sternum. The x-ray picture (Fig. 4) revealed the heart to be enlarged to both sides, but especially to the right.

DISCUSSION

It is noteworthy that while auricular fibrillation and flutter are often found in adult cardiac patients, they are rarely reported in infants

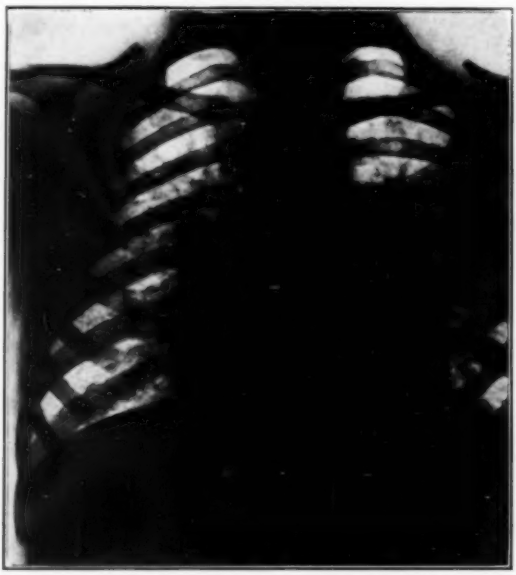

Fig. 4.—X-ray after restoration of normal rhythm; heart markedly enlarged, principally to the right.

and children. This holds true especially for auricular flutter. While auricular fibrillation is not exceedingly rare in children (Schwartz and Weiss[1]), only three authentic cases of flutter in infants have come to our knowledge from the literature on that subject.*

Carr and McClure[2] and Sherman and Schless[3] have described auricular flutter in newborn infants. In both cases the flutter soon changed into the normal sinus rhythm. Amberg and Willius[4] reported the case

*The case of Poynton and Wyllie (Lancet 2: 371, 1926) should be regarded, according to our view, as paroxysmal tachycardia. The electrocardiograms of O'Donovan (Lancet 1: 607, 1929) are not clear examples of auricular flutter.

of a child of two and one-half years who appeared to be suffering from a patent foramen ovale and showed auricular flutter the last nine days before death.

Thus ours is the fourth established case in literature, and it is, moreover, a remarkable one, owing to the fact that the flutter had lasted continuously for nearly three years before it was made to disappear by digitalis medication.

We are not able to say whether the flutter rhythm, as is usually seen under the influence of digitalis, first passed into fibrillation before the sinus rhythm developed. In any case this stage must then have been of very short duration.

Fig. 5.—Patient after successful digitalis therapy (January, 1939), now 5 years old.

The cause of the auricular flutter in our case cannot be positively established. In adults the principal causes are mitral valvular defects, coronary sclerosis, and hypertensive heart disease. There are no arguments for the assumption of acquired heart disease in this case. The child did not have acute rheumatic fever. What seems to us probable is that she has a congenital heart defect, i.e., a patent foramen ovale, the more so as Willius and Amberg[4] have described a similar case. In our case the murmur and the shape of the heart tend to this diagnosis, for the shape, especially, points to an enlargement of the right half. Lastly, it is a well-known fact that this heart defect often terminates in auricular fibrillation and flutter.[5]

Until now the normal rhythm has continued. The child takes a small dose of digitalis intermittently. The general condition is very satisfactory as appears from Fig. 5.

SUMMARY

A case of auricular flutter in a child 2 years old, which continued for three years, is described.

Administration of digitalis was followed by a definite recovery, a normal sinus rhythm resulted, and the symptoms of cardiac failure disappeared.

The child is probably suffering from a patent foramen ovale.

As far as we have been able to discover, this case is the fourth described in literature and the second cured by digitalis.

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THE ROLE OF THE PEDIATRICIAN IN MENTAL HYGIENE

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IT IS now an established fact that the later adjustment of the adult to the social group is largely dependent upon the previous development of satisfactory behavior patterns in early childhood. This being the case, it seems particularly appropriate to discuss the part which the pediatrician might be expected to play in the preservation and improvement of the mental health of his patients.

Since the mental hygiene movement has yet to celebrate its thirtieth birthday, it is rather significant that a number of pediatricians, more prominent among whom are Bronson Crothers and Borden S. Veeder have, for approximately two decades, stressed the fact that adequate medical care of the child cannot be given without intelligent attention to any intellectual and emotional difficulties which may be present. This belief was officially endorsed in 1930 at the White House Conference on Child Health and Protection by the Subcommittee on Psychology and Psychiatry in Pediatrics, of which subcommittee Dr. Crothers was the chairman. Although already interested in the psychiatric side of pediatrics through stimulation by my associates in the pediatric department in which I interned, curiously, my final decision to leave the practice of pediatrics and seek special training in child psychiatry was reached following a conversation, not with a psychiatrist, but with a pediatrician, Dr. Frank C. Neff.

While the truly successful practitioner of medicine has always more or less intuitively considered the personality of the patient in relation to the physical disorders which present themselves, pediatricians generally are rapidly becoming aware that this is not enough. They are developing an increasing consciousness that they must do something concrete about the emotional problems present in many of the children they are called upon to treat. This demand of the pediatrician to exercise his potential function intelligently as a mental hygienist has placed him in a perplexing situation, for in many instances he feels himself inadequately equipped with the techniques of the psychiatrist and the clinical psychologist. He recognizes that he cannot afford prolonged intramural training in child psychiatry, and, when he has attempted to improve his armamentarium through reading in this field, the pediatrician has found himself frustrated by the often confusing technical verbiage of psychiatric literature. Fortunately, leaders in the psychiatric

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field are rapidly taking steps to remedy this situation. While to cover the field of child psychiatry adequately in its relationship to pediatrics would require a series of discourses, it is my purpose to outline briefly some of the more fundamental psychiatric concepts and suggest the manner in which they may be utilized by the pediatrician.

During the last four decades various schools of thought have made their important contributions to the study of personality development. However, there has been a pardonable tendency on the part of most of these groups to stress their own particular philosophy, while minimizing or ignoring the importance of various other factors. Probably the leading psychiatric philosophy dedicated to the consideration of the individual as a whole is that of the psychobiologic school. The psychobiologic principle, simply stated, means merely that in order to understand the personality of the individual, one must consider him as he is physically, intellectually, and emotionally constituted in relation to his total situation, past and present. The adoption of such a breadth of viewpoint as this would seem essential if one is to understand the human personality in health or disease.

In order to remain mentally well, the child within the limitations of his physical, physiological, intellectual, and emotional constitution must be able to adjust to the limitations of his environment, yet at the same time obtain the satisfaction of his basic needs from his relationship with that environment. In other words, he must be able to maintain the integrity of his own personality while meeting the constantly changing situations of life.

These basic needs, the satisfaction of which is so highly important to the well-being of every child, are principally three in number: first, the need for *security*, which is normally provided by the knowledge that one is wanted and accepted by his family and social group and that he need not fear physical privation, destruction, or desertion; second, but equal in importance, is the need for *affection* and *understanding*, which in many ways is a complement to the security of the child, but implies an even deeper relationship of the individual to his family and social group; third, is the need for *achievement* and *recognition* whereby, through his efforts, the person is able to obtain in some measure a feeling of superiority, or at least equality, in his social group and with his particular sex. As long as these basic needs for security, affection, and recognition are being truly satisfied, the child, as a rule, is happy and mentally healthy.

However, because of certain stresses or inherent weaknesses, the child may find himself frustrated in his attempts to obtain the satisfaction of certain or all of his basic needs, and when this happens the child tends to lose his emotional equilibrium. The inherent weaknesses or the stresses increasing the vulnerability to frustration may have been present in very early life, or they may have come later.

Certain inherent weaknesses may come from within the individual himself or may have been passed on to him by his progenitors. While we do not fully understand all the influences of heredity, we do know that the child may inherit a deficient intellectual endowment or the tendency for the development of certain disorders of the central nervous system, such as convulsions, which may handicap him in meeting the demands of life. Furthermore, he may inherit a tendency toward a particular body structure and physiology, which may affect the manner in which the child will react to his life situation. An example of this is the child with an apparently inherent instability of the autonomic nervous system who either underacts or overacts physiologically to external or internal stimuli. He may have suffered an injury at birth resulting in a physical deformity or actual brain damage, forever limiting to some extent the ability of that child to adapt himself to the vicissitudes of life. Congenital syphilis or meningitis may directly affect the central nervous system and reduce the intellectual capacity; or encephalitis or brain trauma may not only result in some degree of intellectual retardation, but also may change the child's pattern of reaction. He may have suffered a relatively incapacitating physical illness, such as rheumatic heart disease, infantile paralysis, osteomyelitis, diabetes, or tuberculosis, which he feels is serving to frustrate him in his attempts to obtain his normal satisfaction from life. Or he may have strabismus, harelip, a clubfoot, large and unduly prominent ears, a seriously disfiguring malocclusion, unusually small genitals, excessive adiposity, unusual thinness, extremes of height or shortness of stature, or some other physical abnormality which makes him feel inferior to his associates and therefore causes him to believe himself less acceptable to his social group. Defects of vision or hearing may often serve to frustrate the child in his efforts to attain equality of achievement in the classroom or on the playground.

Incidentally, overprotection of a child during a protracted illness, or unduly shielding a child who is physically handicapped may often result in that child's resorting to complaints of physical illness or to using his handicap in an effort to avoid meeting the responsibilities of life.

From the psychological standpoint, guilt over erotic wishes, masturbatory activity, or other sex play may produce great anxiety and fear, resulting in serious conflicts which serve to frustrate the child in his adaptation to life. Also, of great importance, is the usually groundless fear of the child that he is lacking in the particular traits which are supposed to be characteristic of his particular sex.

Other very important frustrations may arise from within the home. Rejection by either or both parents, loss of a parent or other loved one by desertion or death, without adequate compensation is likely to result in great insecurity—particularly if these things occur in early childhood. Frequent harsh treatment or ridicule by a parent or sibling, in-

consisteney in discipline, or excessive restriction by either parent will serve to blight the emotional development of the child. Naturally, in the early years, the mother is more important to the child of either sex than the father. Unconsciously, the young child desires complete possession of both parents, but particularly the mother and, not infrequently, may show some indications of jealousy because the parents seem to have a depth of relationship which he feels he does not share. This is quite natural and entirely normal, unless factors develop which serve to intensify in either the little boy or girl a desire for complete possession of the parent of the opposite sex and to exclude the parent of his own sex. This unconscious possessiveness occurs fairly frequently, and the resulting feeling of anxiety and guilt may produce deep-seated conflicts which, if not resolved, will seriously affect the later development of the individual. Because of the child's desire for complete possession of the parent to the exclusion of all others, his security is naturally somewhat threatened by the birth of another child. This is quite a common experience, and in a family where there are wholesome, well-adjusted, understanding parents, this situation usually takes care of itself. However, if the parents themselves are maladjusted and there are indications that some other brother or sister is better accepted by either parent, the child concerned will usually react with the development of hostile phantasies or behavior toward the other sibling and the particular parent involved. Immorality, chronic alcoholism, or other disgraceful conduct on the part of either parent, or the parent substitute, which makes the person less satisfactory as an ideal, may seriously frustrate the child in his attempt to identify himself with that individual.

All of these distortions of the family-child relationships may produce serious consequences, and some have occurred in the life experiences of every maladjusted child or adult. This latter point being worthy of additional emphasis, let me remark that of all the factors which will insure good mental health in later life, probably the most important is the maintenance of emotionally wholesome family-child relationships in early life, this being equal or sometimes greater in importance than the physical constitution and integrity of the individual. Practically every neurosis, practically every case of delinquency can be traced back to an emotionally unwholesome home environment.

Still other significant stresses productive of frustration may come from the environment outside the home. Scholastic or social demands which are beyond the innate capabilities of the child may produce a serious warping of the personality, as may also the attempts of an educational system to force a child of very superior intellectual capacity into a rigid academic mold which was meant to meet the scholastic needs of the student of average ability. One should not overlook the frustrating effect which racial prejudice may have upon a child of a

minority group who seeks acceptance by, or must associate at school or play with, a majority group of an antipodal racial stock. In a discussion of environmental factors affecting the personality, one should not fail to consider the deleterious effect of undesirable associates or the inadequacy of the environment in providing for the constructive use of leisure time.

As many of us now realize, the child is by no means merely a miniature of the adult, for children differ widely in their thinking, feeling and emotional reactions, just as they differ in their reactions to physical disease. This must be kept in mind at all times by the physician dealing with children as patients. To the developing child, his world—filled with mystery and awesome in its proportions—is constantly expanding before his eyes, arousing in him an insatiable curiosity and seriously trying his powers of comprehension. At times, this expanding world threatens to overpower him by its immensity so that he is greatly dependent upon the adults with whom he is associated for explanation, reassurance, and protection. Feeling the need to master his world, he strives to become all powerful like his mother or father, but, as natural growth is too painfully slow to meet his immediate need for omnipotence, the child bridges this gap by the invention of many wishful phantasies, in which he always emerges victorious. Sometimes when the child's adults seem to fail him, his world appears at any moment to sink beneath his feet and engulf him, causing his little fears to become magnified and multiplied. It is these fears, tending to dominate his waking phantasies, which carry over into his dreams and result in night terrors and somnambulism.

Just as the child is more a creature of feeling than of thinking, so also he differs from the adult by being often unable to relate correctly an unpleasant experience to its remote but logical cause, or to comprehend the often annoying delay between his expression of a wish and its ultimate gratification. Nevertheless, the child has a keen but rather circumscribed sense of justice. Because of his limitations in comprehension and judgment, the child is a subject of impulse rather than deliberation. He tends to seek the prompt gratification of every desire and the immediate expression of every revengeful wish, either directly or indirectly. While these attributes of impulsiveness and delimited comprehension and judgment are often found in the emotionally immature adult, they are normally present in the child.

Not infrequently adults erroneously believe that things the child observes in his early years are incapable of leaving any lasting impression. While the young child is not capable of comprehending affairs from the adult's viewpoint, this very immaturity often results in serious misunderstandings and misinterpretation in the mind of the child of things which go on about him. Very important in this respect are

the misimpressions which the child may receive from observing the marital act between the parents, serious quarrels between parents, or the poorly explained prolonged absence of one or the other parent from the home.

After this brief discussion of some of the constitutional limitations and environmental factors which may influence the child's mental health, it might be well to portray the character of the symptoms of mental illness and what the maladjustment means as far as the child himself is concerned. The symptoms are merely a perverted expression of the unhappiness which the child consciously or unconsciously feels. Mental illness or behavior disturbance is in a large measure just a disguised expression of the unhappiness and dissatisfaction with the situation in which the patient finds himself, superimposed upon which there will be in the cases with organic brain changes the alterations in function one might expect from the actual destruction of nervous tissue. Let me emphasize the fact that the reaction of the child to his unhappy state is not a conscious one, for he usually little understands, and may be totally unaware of, the factors which are responsible for his condition.

The unhappiness may perversely express itself by varying degrees of disturbances within the child himself, by the development of excessive shyness or timidity, by daydreaming, or by the appearance of such symptoms as enuresis, urinary retention, night terrors, somnambulism, paroxysmal tachycardia, essential hypertension, dysmenorrhea, amenorrhea, menorrhagia, or by disturbances of the gastrointestinal tract such as anorexia, persistent vomiting, chronic constipation, recurrent diarrhea and spastic colitis, with even mucous colitis being the end result at times. While any of the preceding symptoms should be diligently studied for any organic factor which may be responsible, one should not lose sight of the fact that all of these conditions can be produced on a purely psychogenic basis.

The unhappiness of the child may unconsciously express itself by alterations in the relationship to his family group by the development of excessive dependence, stubbornness, temper tantrums, destructiveness, stealing, truancy, or other hostile behavior. Also the child may unconsciously express his unhappiness by alteration in his normal relationships with society, such as by the development of bullying, assaultiveness, shiftlessness, vagabondage, sexual perversions, dishonesty, thievery, and even homicide.

Few physicians consciously recognize the role which they are forced to play in their relationship to the child. Because of his own experiences and needs, the child unconsciously identifies every new adult whom he meets with some other adult with whom he has been more closely associated. For example, the child identifies the nurse or teacher with his own mother and desires to establish with that individual the relationship which he would like to have with his mother. Also, in rela-

tion to that mother-substitute he may unconsciously give expression to hostile impulses which he does not dare to express toward his mother. This accounts for much of the misbehavior in school children who are relatively submissive in the home. Quite logically, in the mind of the child the physician usually becomes identified as a father-person. This places a heavy burden of responsibility on the pediatrician. If he ever expects to win the confidence of his little patient, the physician must comprehend the world of the child, provide him with adequate opportunity for self-expression, and approach him always with an attitude of sympathetic understanding and acceptance.

Every father-person serves a dual role in the life of the child. He is at times the "good father" who gratifies his wishes and makes the child secure from the onslaughts of his environment. However, at other times he is the "bad father" who punishes the child for his transgressions and restricts him in the immediate gratification of some of his impulses. As the physician must, at times, cause the child physical pain or embarrassment, he must be cautious lest through haste or brusqueness he become the "bad father" rather than the understanding, tolerant "good father." Usually the child will tolerate considerable discomfort and pain if he first fully understands the nature of the procedure, its necessity, and the goal to be achieved thereby, and if he is firmly convinced that the procedure is not a perverted form of punishment for his actual transgressions or aggressive phantasies.

Because of the taboos and mystery with which sex has been surrounded, the sex organs commonly assume an undue importance in the mind of the child. Hence, any operation upon the genital region may be attended by the development of serious fears and conflicts unless the operation is previously explained to, and comprehended by, the child. Parents too often threaten to amputate the genitals of the boy as punishment for masturbation, or severely whip the little girl for rubbing her genitals or wetting her bed. The physician, too, should also be cautious about the too frequent manipulation of the genital region of the child of either sex, for all too often the child may develop such an extreme attachment to the physician that it may result in incestuous wishes of the child toward the father-substitute and interfere with his later adjustment.

Since the child seeks to understand all he observes, it is only natural that he should be curious how the new baby appeared out of the apparent nowhere and why his body is different than that of his sibling or playmates of the opposite sex. This curiosity on the part of the child is entirely wholesome until made vulgar by the attitudes of the adults in his surroundings. Every child should be given a wholesome explanation of these mysteries at the time when his curiosity first expresses itself. Ideally, this information should be imparted by the parent who may at times counsel with the physician. However, at times the parent

is either unsuited or unwilling to do so, and the physician will be faced with the necessity of conducting this part of the child's education. No matter how immature they may seem to an adult, queries in the mind of the child are always of great importance to him. Consequently, the questions of children should always be given serious consideration and as clear, truthful, and understandable answers by the parent or other adult as possible. It does no harm to the prestige of an adult to tell the inquiring youngster that the answer to the particular question is not known. Mention should here be made that children of either sex sometimes develop the fear that their sex practices will result in physical weakness, intellectual impairment, or insanity. Of course, these fears, which are groundless, have usually been implanted by some adult, usually through ignorance. When confronted with such a situation, the pediatrician should take great pains to see that the child's fears are allayed and that he assumes a more wholesome viewpoint regarding the matter.

Of all the branches of the medical profession, the pediatrician is in the most ideal position for accomplishing the maximum of good from the standpoint of mental hygiene. Not only does he have a great natural ability and special training in handling children, but also he often sees the child when a potentially serious emotional disorder is still in its incipiency. Then too, the pediatrician, being a permanent resident of the community, is likely to have at his finger tips a great deal of valuable information concerning the home life, the past experiences, and the environmental situation of the patient which is invaluable in obtaining a clear picture of the total personality of the child. Furthermore, because of the permanent residence of the pediatrician, he can follow the child with a personality disorder for several years, if necessary, with comparatively little expense accruing to the parents.

Since the foundations for the personality disorder in any particular case may be very complex, technical assistance may often be necessary as an aid to evaluating the total personality or potentialities of the child, or to arrange for certain manipulations of the environmental situation to aid in the more speedy recovery of the child. For example, it may be highly important to utilize the services of a clinical psychologist to ascertain, not only the intellectual capacity of the child, but his special abilities and disabilities as well. The services of a well-trained social worker may be needed in providing financial assistance to the family, and in some instances the social worker may be able to take the time, ill-afforded by the busy pediatrician, to alter the attitudes of the child's parents or teacher. The social worker may also be of assistance in bringing about a modification of the school program to suit the capabilities and needs of the child, or she may arrange for the child to participate in a suitable program of supervised leisure time activities when that is needed. The psychiatrist, with his special techniques, may be

needed to go more deeply into the emotional life of the child so that the information thus gained can be used by the pediatrician in the future management of the case. These three technical specialists—the psychiatrist, the clinical psychologist, and the psychiatric social worker—are found working in a cooperative relationship in a child guidance clinic to which the child may be referred by the pediatrician if he so desires. In the same manner the services of the endocrinologist, the ophthalmologist, the neurologist, the roentgenologist, or biochemist may be needed so that the pediatrician will have a more comprehensive understanding of the human material with which he has to deal.

Since all of us have our limitations, the pediatrician can at times be of great assistance to the patient with a personality disorder if he recognizes that the trouble is more serious in nature than his time or techniques will permit him to treat safely and if, upon recognizing that fact, he does his utmost to persuade the parents to accept the needed treatment of the child by a psychiatrist and to receive that treatment in the protected environment of a good mental disease hospital if necessary.

SUMMARY

I hope that from this may be obtained a clearer realization of the complexity of the factors which may be productive of a personality disorder and which therefore often make a cooperative effort necessary in the study and treatment of children with such personality deviations. In every case of a personality disorder it is necessary to consider the child carefully from the standpoint of his physical and intellectual constitution and his emotional life in relation to his total environment, past and present. The early recognition and treatment of behavior disorders and emotional conflicts in children can do much towards averting more deeply rooted and serious emotional disturbances in later life. Because of this, it should be apparent to all that the pediatrician has a very important contribution which he can make to the problem of mental health.

Special Article

FAMILY RELATIONSHIPS

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THE fact that many volumes have been written on the subject of family relationships bespeaks the importance of the subject, but also makes difficult the assignment of adding any fresh point of view. But a series of articles on the mental health of the child that omitted this large subject would leave untouched influences which vitally determine the quality of the child's personality. This brief statement will have as its purpose the evaluation of some normal aspects of family life in our present culture and will point the discussion around the needs of a growing child and how they are satisfied. The needs of the child for healthy development are apparent. He needs a sense of belonging and to achieve through living a feeling for his own inner value. How he achieves these and other essentials for health requires a discussion of the balance and interplay between the parents' responsibility and capacity to satisfy these needs and the emerging desire and ability of the growing child to be responsible for meeting these needs with his own developing capacities.

As a preamble to the discussion of this subject, I want to draw attention to certain evolutionary aspects of family life and relationships. The child as he grows has not only a relation to his family but to the culture within which his family is a small but integral unit. In different periods of man's history, and in different cultures of each period, there have been many variations of family structure and function. So wide have been the variations that the term "family" is not easily defined. In one culture it may refer to a small group bound together by the convention of marriage and maintaining a separate unity within a larger social organization. In other groups the term may define a more loosely organized group bound together by economic rather than biological factors. In some cultures there may be the dominant matriarchal influence, and in others the patriarchal influence may be in the foreground. Some family units attain the status of a clan, and the element of kinship binds together a considerable group of people who try to maintain a completeness within their group that in other cultures is found in the interrelations between the family and the larger social unit within which it functions.

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During the historical evolution of man and family life there have been many shifts in its form and function. But through these shifts there has run an important common dilemma. The family constitutes the smallest social unit within a larger unit. There have always existed varying degrees of competitiveness between the family and clan for the primary loyalties of its members. The family has always touched the deepest feelings of mankind because here man and woman live in closest contact with each other. Members of families in their living relations supply each other with a completion of their living selves in a way that is unique. The child is the product of two people and becomes not only a new person, but a symbol of their togetherness. But the clan or nation demands some of that loyalty. The safety of the family has required the larger organization which the clan or state provides and that has been maintained through a larger and a different feeling of belonging.

As cultures became more complex, the needs of its members required new tools for their satisfaction. The family slowly ceased to be a self-sufficient unit but began to depend upon other resources for a better performance of certain functions. Going along with these shifts, the clan or state in order to gain more support began to take over many family functions. Family change has been associated with the growing demands of the larger unit. We have seen, therefore, the family becoming less important as a center for education, for religious instruction, for companionship and recreation. There can be no doubt that in some cultures there have been open attempts to destroy the family and to start the child, almost from birth, with the feeling that he belongs to the state and not to any smaller division. But such attempts only serve to focus attention on certain indestructible aspects of family life—influences which have always operated and which have made the family, in one form or another, almost synonymous with human existence. Where there is life, there is a relationship to another; where there is a child, there is a mother and a relationship together, usually in a family setting.

Thus these shifts in family structure and function must be viewed as change rather than signs of deterioration. In every period there have been those who have predicted the break up of the family. But it continues, and will always be a part of life. But these shifts which have gone rather rapidly in an age of rapid transit, radios, moving pictures, etc., have made it necessary to redefine the essential function of a family and how it meets some of the basic needs of its members, particularly the child.

These shifts have served to focus attention upon the basic unchanging and everlasting function of a family: where people live together, where they experience their most fundamental feelings, and where a child gains his first sense of self in his relations to others. It is the mutual experiencing of feelings that establishes the bonds that make the family

unique and stirs loyalties that can never be fully transferred to a larger and less personal relation to a state or clan. In those cultures which seem to have the most stability, these loyalties are not in competition with others. Rather, they supplement each other and strengthen the individual who can make creative use of his sense of responsibility both to family and to state.

We live in a culture that still respects the individual as the foundation stone of the social order. A vigorous and healthy individualism is almost a basic requirement of a creative society. It rests upon a belief and respect for difference which is the essence of individualism. A healthy individual is one who believes in his own uniqueness and difference, which belief leaves him free to respect difference in others. If this is a base for a healthy society, it must be the base for a healthy normal family group. So I would place that first as the major contribution of family life. Here individuals with different experiences and backgrounds learn to live out their differences in a relationship that is close and binding. They can find their satisfactions in a sharing relation rather than in one where one self engulfs and submerges the other. Man and woman participate in the creation of a child and have the important and fascinating responsibility of guiding the growth steps of a child as he achieves through living a self that is his own and different from those who create and direct him.

The philosophy that people have about difference is the determiner of family relations and the influence necessary for mental health. Such a family, whose members have a live and let live attitude, can provide a framework for the differentiation of its members. By contrast, this becomes clear when we think of an atmosphere dominated by the drive for self-immortalization, where a person would seek to live forever by perpetuating himself in another and trying to make of a child a rubber stamp of himself.

When man and woman have acquired through their own development a healthy feeling of their own individuality, they are in position to create a child and guide him to maturity. They can give all that must be given to the child of their creation without trying to perpetuate that feeling of ownership which can block the child in the achievement of his own independence.

It seems worthwhile to devote the balance of this paper to a more careful consideration of those family relationships which are built on a healthy respect of the individuality of its members and how this philosophy can influence the relationship between parent and child.

A child needs a great deal from parents. His life depends on what they give. His physical life depends upon the food and shelter they provide and his emotional life is conditioned by the quality of affection and direction they are free to give. I would stress here the freedom to give, because of the need of naturalness and spontaneity in the giv-

ing. A parent cannot be told how to love a child, nor can he be given the rules and regulations of being a good parent. Children need real people for parents and not intellectual artifacts. The parent who knows all the answers on how to be a parent is sometimes a person who cannot trust his capacity to live out that knowledge in his everyday relation with the child. Those individuals who have found through their own development a feeling that there is value in being what they are will be those who can give freely and naturally to a child. Methods become the expression of their own realness. They can modify method through more knowledge and become more real in the living out of their responsibilities. But knowledge alone never made a good parent.

Parents will always have a sense of responsibility for a child they have created. What he is at birth is what they have made him. Truly, a child is an extension of themselves, a feeling that has more living significance for the mother. But growth necessarily begins to change this feeling because through each experience the child has he learns his own separateness and gradually acquires a feeling of being responsible for what he is and what he can become. Through these changes the parents begin to experience a change in their earlier feeling of complete responsibility. As they can let that feeling be broken up, they are allowing the child opportunities to feel responsible for himself. This is the process of differentiation, the success of which is so basic for mental health.

This feeling of responsibility that one human has for another has been stressed in such a way in literature that many parents have been overwhelmed by the seriousness of their task. Certain unhealthy sequences have followed when the parent tries too consciously and too purposely to live out this responsibility and be a good parent. Certain common problems illustrate the dilemmas created by the overintensity set in motion. For example, affection is a feeling that needs a spontaneous and natural expression. Children need that. But when a mother, aware of the powerful influence of affection, begins to parcel it out in carefully measured doses to prevent a "mother fixation" or to avoid the horrors of a "spoiled child," she is introducing an unnatural element into the child's life. She is taking her responsibility too literally and too completely. It is probably true that just as many children are confused and disturbed by the parent's fear of spoiling as are upset by the possessive and controlling use of "affection," which does create a spoiled child. The parent who tries too hard to reduce his affectional expressions to rules and techniques fails to appreciate the capacity of a growing child to make responsible use of the natural and spontaneous expression of feeling which characterizes the healthy parent. Parents who can give such real expressions of their feeling can be equally free to maintain a growing feeling of separateness to the child of their creation. They can continue to build their own adult life and interests and still maintain their part in the life of a growing child. This is the natural

breaking up of the more complete feeling of responsibility during infancy, but necessary in a different and less complete way as the child gains, through the use of what a parent has given, the courage and capacity to move toward separate living. Parents who can accept the responsibility of being persons in their own rights can encourage the necessary separation and differences which growth requires. They can feel increasingly that the life they were responsible in creating can continue through the gradual breaking up of that more complete feeling of being necessary for all that a child is and does. They can use their relation to encourage a child to find the self that will be his and which will have the important qualities of difference.

A growing child needs not only affection and a feeling of belonging in his relation with parents; he needs also the comfort of authority. He needs a person who, in being real, can limit the emerging power a child comes to feel as his own. The child needs limits to this emerging strength in order that he can acquire an organization that allows for its creative use.

Here again there has been a good deal of confusion, made more articulate by some well-intentioned but not always wise efforts to "train" parents for the great responsibility of their task. Discipline is a word that has a slightly old-fashioned connotation in an age that always seeks to be reasonable about everything. The doctrine that states that a parent should always explain requirements to a child is a doctrine that is sometimes as confusing to a child as it is to a parent.

A growing child needs not only affection and a sense of belonging in his relation with a parent; he needs, and even craves, the comfort of authority. He needs a person who, in being real, can define limits to his emerging strength—limits that are not designed to crush and overcome this strength but to give direction and creative value to it. If every limit has to be explained and its purpose defined, it frequently ceases to have any realness because of the discussions set up. A child can struggle against the parents' conceptions of reasonableness more effectively sometimes than against the original requirement. If he is in a struggling mood, he can find as many arguments against going to bed as the parent can find for it.

Probably the more important force in this area of discipline is the parent's feeling of the right to be direct and to require some conformity to a world that requires both assertion and yielding. Where there is a basic respect for the individual being directed, some things can be required without lengthy explanations and because a parent wants something done. A child has the right to rebel and a parent has the right to stand firm. But it does take more courage to be that direct. It does require belief in one's self tempered and colored by belief and respect in the child who is being helped to live in a world that does not give way to rebellion and whim.

Where the relationship rests upon a healthy respect for difference, which follows when one is oriented to a relative set of values about responsibility, a discipline that has creative values will usually follow. A child is being brought up in a world that is not always going to be reasonable; therefore, a parent who feels so responsible for a child that he tries to protect him from any discomforts is really saying that he cannot believe the child has anything in himself to deal with uncomfortable realities.

This is not a defense of unreasonableness in direction. It is more an attempt to say that reasonableness and fairness become a part of a living relation when there is not a too conscious and purposeful attempt to achieve it. Helping parents to a natural belief in their own convictions usually allows reasonableness to be a part of their relation with a child. Their ability to be real in their relationships allows for realness to emerge in those who are dependent upon them.

In summary, pediatricians and psychiatrists, who have the professional responsibility of clarifying for parents and children the nature of their relationship, can be more effective as they are clearer about the basic nature of the relationships involved. This statement has sought to emphasize that healthy human relationships grow from a respect for the individual differences. A healthy parent is one who has gained through living the courage to be himself, which allows the emergence of a feeling of unity with others whose differences he accepts. He respects the value of what he has to give to a child and, at the same time, believes in the capacity of the child to make use of what is given as he moves toward a more complete feeling of his own separate individuality.

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Round Table Discussion on Child Psychology
Early Symptoms of Behavior Problems to Be Met in Pediatric Practice

Chairman: Dr. Mary H. Layman, San Francisco, Calif.
Assistant: Dr. Horton Casparis, Nashville, Tenn.

CHAIRMAN LAYMAN.—Let us please have a frank exchange of experiences this afternoon. Since child psychology is a fairly new subject, suppose we open with a review of the background.

Growth and Development

Mary H. Layman, M.D.

At the first session of the Academy of Pediatrics in 1931 the report of the mental hygiene committee under Dr. Bronson Crothers as chairman met the challenge of rapidly expanding child guidance work with discrimination and vision. I quote: "The report is one of confusion and progress, and the main point I would like to make is that if child guidance is not a part of pediatrics, I don't know what it is." Since then child psychology in some phase or another has been represented on the annual programs of the Academy, and various members have gone far in the past few years towards the realization of that longed-for ideal, namely a better fusion of the knowledge so far accumulated in the sciences of pediatrics, psychiatry, psychology, and sociology. A couple of years later, at a round table discussion in Toronto, Dr. Blatz remarked the following: "I would guarantee, that in ten years, if this group meet here together, the type of discussion, that we would hear, providing they learn, will be so different from this that it will be as the difference between a kindergarten and a graduate school." That was only five years ago, so I shall assume that we are today at the high school stage with great hopes for further improvement.

The reason, which compels me to hark back to the beginnings of psychology is that we still find in many of our contemporaries' minds, either on the surface or in the subconscious strata, a large and interesting collection of notions dating back to at least the middle ages. These more or less handicap effective approach, treatment, and outcome of the problems confronting us now. Human nature demands the finality of an axiom and holds what it considers such with an unrelenting grasp; science answers with a question mark, as it can never be final. The basic maxim as expressed by Chilon in the sixth century B.C., "panta rhei"—"everything is in a state of flux," is as true today as it was then: knowledge means constant learning; judgment means constant revaluation; life means constant readjustment.

The initial antagonism to the introduction of anything seemingly so foreign to the field of pediatrics as psychology finds its *raison d'être* in the general conception of psychology as the academic science it was in centuries past. As recently as one hundred years ago, more or less, psychology was still closely allied to philosophy and like it a speculative science, evaluating human behavior on the basis of moral standards. The analysis of man's nature was not based on what man actually was, but on what man was supposed to be, and his reactions judged not by what he did, or could do, but by what he should do. Our homes, our school system, our courts, our government are only just beginning to emerge from these medieval attitudes. Not without their readjustment and the general acceptance of a new attitude can we boast of any real progress in human relations, which, after all, is the ultimate goal of psychology.

During the last century an overwhelming number of scientific discoveries ushered in an era which saw fit to relinquish abstract thinking in favor of concrete and tangible facts. At this time psychology emancipated itself from philosophy under the leadership of Wundt in Leipzig and borrowed the laboratory and its tools from physiology, predominantly from the physiology of the senses. Paralleling the physical experience ran the mental reactions of the subject, that is his own observations of feelings and thoughts which accompanied the experiment. This was the introspective stage of psychology, which, though experimental, suffered from the bias and fallacies invariably inherent to self-analysis.

Meanwhile chemistry, physics, and biology were taking the decisive strides, which ultimately opened up a most fertile field to psychology, a field of which we have but barely scratched the surface. The same experiments by which Darwin and Mendel laid the foundation for the theory of evolution and heredity of physical traits served for a like logical sequence of heredity in mental traits.

No longer was a man blamed for not behaving as he should, but rather was regarded with awe for what he could not help but be: heredity held sway. Nevertheless, the individual, even as a twin, did not run completely true to form, but showed modifications obviously wrought by the environment. A certain group of people never have accepted this theory for mental traits and still persist in seeing human beings born as perfect mental blanks to be developed, molded and fashioned by postnatal experience alone. Thus is kept alive the discussion of the balance between heredity and environment, while research is almost daily adding to the support of either one or the other.

Based on Mendelian principles, the convenient assumption of unit characters led to a useful, if oversimplified, method of classification and measurement as outlined in so-called mental tests, the actual number and variety of which belie the uniformity of what is measured and what is assumed to be "mind." Out of the confusion which reigned in such groups as educators, judges, and psychologists arose an appeal to Binet, whose keen intuition was able to discern in the maze of mental manifestations a fundamental organization with inherent definite characteristics and—within the limits of certain variations—closely linked with the age of the child. His test, brought up to a remarkably discriminating scale by Terman, is almost universally used, and, like the thermometer and microscope, will remain an indispensable tool in the hand of the psychologist. It measures *basic* ability, not special talents of dexterity and, if properly interpreted, serves as a background for the shape and form required for further approach or treatment. For instance, to use an illustration and assuming average conditions, I can, in order to demonstrate a good memory, make the child repeat numerals; at 2½ years 2 digits, at 3 years 3 digits; at 6½ years 4; at 7 years 5; at 9 years 4 reversed; at 10 years, 6 straight; at 12 years 5 reversed; and as superior adult, 8 or 9 straight. Or I could obtain favor with a child of 2 years by presenting a tongue blade, of 5 years a candy, of 10 an airplane, of 14 a football outfit, of 18 a book,

and beyond that a job perhaps. The *purpose* and the *response* are the same in every instance (desire to elicit and achieve the demonstration of a good memory and currying favor and obtaining it), but the *form* is adapted to the basic capacities of the individual. Were I to change the sequence, I would be ignoring the respective basic capacities and court failure instead of success. Naturally I am using the concepts of "memory" and "favor" very cautiously and only for convenience' sake. I know full well that science—to make confusion worse confounded—proceeded to shake the foundations of physics by blasting the atom, while similarly blasting the unit character into countless bits. If, as it is now assumed, it takes at least 50 genes to make up red in the eye of the fruit fly, how many genes would go to make up "memory"?

Provided that psychology could detect all the basic abilities and education knew how to exploit them, still these attempts at guidance would be thwarted by the most powerful forces of any that shape our actions, the emotions. Though psychoanalysis, through its principal exponents, Freud, Adler, and Jung, has opened tempting vistas of unexplored territory, most of us remain hesitatingly on the outskirts of what looks like a dangerous bog; a few courageous ones are laying planks across to allow them to peer into the unknown depths without soiling their feet. The psychoanalysts claim—no doubt with authority—that they have developed means and methods by which to drain this bog, which should and could be a beautiful garden, if only the destructive forces were diverted at the source, the source being the basic emotion, the libido.

Perhaps the greatest objection to psychoanalysis lies in the narrowness of its field with overemphasis on the attitudes and underemphasis on the environmental factors. Moreover, merely recognizing the emotions as the source of complex behavior does not give us information about their origin. So long as we cannot hunt down the antagonist to his lair, he may not be more than held in check temporarily (repression) or at best kept out of harm's way by putting him to profitable work (sublimation).

At this point of the dilemma the laboratory again stepped in and supplied psychology with data based this time on body functions under the influence of internal and external factors: on the one hand checks and stimulations from the nervous system and secretions from the endocrines; on the other hand, physical and chemical forces of the outside world like poisons, temperature, barometric pressure, etc. This stage we refer to as psychobiology.

We know what subtle changes in behavior are brought about by minor difference in the over- or underexcitability of the nervous system or the over- or underactivity of the thyroid or the pituitary. We know what gross changes are brought about through the influence of alcohol or drugs; we cannot overlook the fact that changes in personality like arrogance or lack of self-control in the tropics may ensue after moves from temperate zones; and as regards barometric pressure—at the Summit House on Pike's Peak, it was found necessary to station a deputy sheriff because of the critical tension which took possession of the guests at that altitude.

Divine curiosity in its unquenchable thirst marches on. Even after biology has exhausted its physicochemical field, a psychologic puzzle remains, analogous to the old conundrum: which was first, the egg or the chicken? Once we know the relationship, say of the adrenals and anger: Are we angry, because the adrenals secrete? Or do the adrenals secrete, because we are angry? And if so, what makes us angry? The psychophysical parallelism is outdistanced by the psychosomatic.

And now we come to the last consideration, which brings into the picture the fourth allied science, namely sociology. Suppose we do attain the superachievement of knowing the origin and causes of human behavior and the methods by which to influence them, we should still have to ask ourselves which behavior must

we consider the normal one. Dr. Doll defines normal in the following way: "One has to distinguish what is normal in the sense of average contrasted with normal in the sense of sound or ideal. Abnormal, therefore, is deviation from the average and departure from the desirable." There are times when the average behavior may be desirable; at other times it may be a hindrance or even a detriment, as under great emotional stress, in emergencies, or on a desert island. And again departure from the desirable may be the expression of a perfectly average reaction: when a mentally retarded child is nagged at home and ridiculed in school, he runs away—a perfectly logical and simple solution of an untenable situation, commensurate with the ability of the child but commonly considered an undesirable behavior. Thus desirability of behavior fluctuates with personalities, localities, and situations and is not easily agreed upon. Desirability resolves itself into "a satisfactory adjustment to life." And yet, even so, how often have not *unsolved* conflicts led to the greatest and finest achievements in life!

This is the background against which I would wish you to view the subject we are now presenting. We shall for the sake of a clarity of tendencies, bring out isolated features in sharp relief. It is for you not to be duped by oversimplification, but rather to assign the leading themes their proper places in the dynamic, pulsating symphony of life.

And now to our subject of the evening.

If in my introduction I wished to guard against the possibility of conveying any impression of finality, I now paradoxically wish to emphasize certain basic trends which we have to accept as such and utilize to the best advantage until we discover better ones.

If the pediatrician wants to recognize early signs of failing physical health, he must first know the picture presented by good health; if the pediatrician would recognize early symptoms of mental maladjustment, he must first be acquainted with normal or average behavior. Consequently the child you meet tonight is, in the main, the so-called average child. If I refer to the following behavior as normal, you will remember that norms, after all, constitute only a frame of reference, albeit an exceedingly handy one.

According to Arnold Gesell's schedules of child behavior, which are well known to you, I will demonstrate and illustrate by motion pictures the developmental behavior of the infant as he reveals himself on successive maturity levels in characteristic patterns, the expression of an intrinsic, lawful mechanism. This mechanism continues to operate all through life, hidden by overlying acquired traits in older children, but easily demonstrated in infancy.

Dr. Macfarlane will then show you what happens to the individual endowment under the influence of environmental factors, between 1 and 10 years of age, while Dr. Casparis will complete the stages of childhood and present the adolescent, closing with the pediatricians' vital contribution to the detection of first signs of trouble.

Child Behavior From Birth to 18 Months

In physical growth we recognize that the maturation process follows a very definite sequence. A series of very characteristic pictures, or patterns, appear at set intervals: the first teeth erupt at 8-10 months, the 6-year molars and the 12-year molars erupt at their respective times; tables show height and weight to be expected at certain ages; in adolescence we look for typical signs of secondary sex characters. All these changes occur in well-ordered fashion and at very definite times. It takes time to grow. Growth, physical and mental, is closely and intimately tied to time, and as the organism gets older, it takes more time to grow an equivalent amount of the preceding period; that is to say, the growth increments increase with age. In analogy to Minot's graph for physical growth, which charts equivalent amount of growth in not equal, but proportional, amounts

of time, Gesell assumes mental growth progress to follow the same percentage increment; this is actually borne out by clinical observation. In the early phases of the mental growth cycle the dynamic importance of small units of time is so great that every month witnesses some significant increment of behavior. There is a definite relationship between the units of age and the ordered units of behavior, definite patterns for definite ages. These patterns of genetic sequence insure a basically similar growth career and insure to each species its behavior. If chance played any considerable part in the determination of development, a number of infants of the same age would display a vast array of behaviors, which they obviously do not do. Development is characterized by an orderliness which is surprising; one performance precedes the other at a certain stage of maturity. The development sequence cannot be altered, nor the length of the stages shortened; only innate differences in potentiality alter the rate.

Maturation of behavior goes on in spite of physical handicaps; it is remarkably resistent to adversity and tends to grow in obedience to inborn determiners, regardless of outward influences.

Animal experimentation offers many a proof. Carmichael, for instance, experimented with salamander and frog eggs; he added an anesthetic to the water in which he placed part of the eggs, while the rest he placed in fresh water as controls. These developed in due time and learned to swim at their usual rate, while the anesthetized ones, though growing, remained quiet. However, as soon as they recovered from the anesthetic, they immediately swam about as vigorously as the controls had done after a certain time of development. Sheppard and Breed duplicated the experiment with the chicks, one group of which they fed by tube in order to eliminate practice in picking, while the controls, as soon as hatched, began to pick, first awkwardly, missing most of the grains, but improving with continued practice until they were quite proficient and scarcely missed any; then they allowed the artificially fed ones to start picking and discovered that from the start they were never as awkward as the controls had been and that they reached proficiency in a remarkably short time. These experiments demonstrate that, independent of practice, a maturation process goes on regardless of what seem to be retarding influences. It goes without saying that opportunity has to be offered for this ability to express itself, but the type of response will depend on the time at which this opportunity is offered, not on the opportunity alone. "Inborn propensities assert themselves at ascending stages—training and habituation establish them." In human beings nature has offered opportunities for similar experimentation in the occurrence of identical twins. To these the method of co-twin control is applied: that is one twin undergoes intensive training, while the other is left to his natural development. This training Gesell and his co-workers applied, among other test situations, to climbing stairs. One twin was given training in climbing, while her twin did not climb at all. But, when twin No. 2 was given the opportunity to climb the steps which her sister had conquered in twelve long weeks of arduous work, she climbed up unassisted at the very first try, and within two more weeks was as skillful as her trained twin. A child can do neither more nor less than the maturation and organization of his behavior patterns will permit. The unfolding of this organization is concentrated in the earliest period of life. Training is defeated by the process of maturation. To attempt to teach a child before he is neurologically ready is to work against odds or even the impossible—a waste of time and energy. Worse yet, it may make the child stubborn and negativistic. If teaching would wait upon adequate maturation time, effort and vexation on both sides might be saved, while skill and success and satisfaction would be increased. The duty of the educator, parent, teacher, doctor is to see that the capacities of each stage are fully realized, not that the stages succeed each other in as rapid succession as possible.

I have fully and freely quoted from Gesell's publications on this subject.

Just a few more words in explanation of the pictures.

It is easiest to evaluate symptoms of development when two levels are presented for comparison and the same behavior is visualized in terms of higher and lower levels. I am going to show you in a series of six subjects the most startling as well as the most rapid changes, covering as they do the earliest stages from birth through 4, 6, 9, 12, and 18 months. The groups overlap. In each group, as we proceed, the older child of the preceding group represents the younger child of the following group.

Since my film is silent, it does not include language: the other three major categories are illustrated, namely, motor, adaptive, and some personal-social abilities. Motor development is self-explanatory; by "adaptive" is meant the child's attitude towards things in his surroundings; the personal social category composes his reactions to people.

The subjects for these movies were chosen at random from the attendance at a well-baby clinic, the only stipulation being that the children be physically well and the required number of months old. The pictures were taken under the most unfavorable conditions possible, and yet the results were so strikingly unequivocal that no retakes whatever were required for the demonstration series and cuts for lengthiness were reduced to a minimum.

These schedules, incomplete though they may be, definitely show the lawfulness in development and the characteristic patterns of behavior which we may consider as prototypes for many others and as fundamental for behavior as a whole.

*The Genetic Sequence of Crayon and Paper Behavior**

0-1 mo.	Reflex clasp of crayon without visual regard.
1-3 mo.	Increasing complexity in manipulation reactions to crayon without visual regard.
3-5 mo.	Increasing visual cooperation in manipulation of crayon and paper. Clasps (with two hands) if paper favorably presented. Picks up crayon on contact with hand.
6-9 mo.	Reaches for crayon on sight. Brandishes, bangs, and crumples. Does not bring paper and crayon into exploitive relation. Hand-to-mouth reaction frequently dominates.
9-12 mo.	Gradually brings crayon and paper into productive relation. Makes staccato banging marks; or faint wavering scrawl. Gives fugitive heed to demonstration of scribbling by examiner.
12-18 mo.	(Imitative) scribble. Transient, fitful exploitation by crayon with fugitive attention to marks produced. Increase of controlled innervation in bringing crayon to bear upon paper.
18-21 mo.	More defined and spontaneous scribble. Makes a crude imitative stroke. Differentiates between a straight stroke and circular stroke.
24-30 mo.	Imitates a vertical stroke. Shows prolongation of attention span in crayon activity; at 30 mo. horizontal stroke.
30-36 mo.	Makes two or more marks in imitation of a square cross, but does not make adaptive combination of strokes; crude circle.
3 yr.	Imitates a horizontal stroke. Brings vertical and horizontal strokes into relation in imitation of a cross. Copies a circle from a model.
4-5 yr.	Copies a cross. Copies a square. (Draws a recognizable man.) Begins to differentiate between square and oblique cross drawn for model and triangle.
7 yr.	Diamond copied.

*Shown by moving pictures.

Adjustment to Cube

1 mo.	No regard.
3-4 mo.	Passing gaze.
6 mo.	Grasp, approach to mouth.
9 mo.	Grasp and bangs it on table.
10 mo.	Holds one cube over another, no release.
12 mo.	Searches for and unwraps it from paper folded about it. incipient tower; rudimentary release.
18 mo.	Builds a tower; 4-5 or more.
2 yr.	Assembles blocks in a row.
3 yr.	Builds a bridge (3 cubes) (copy).
4 yr.	Builds a gate (5 cubes) (copy).
5 yr.	Builds a stairway (10 blocks) (from memory).

Pellet Prehension

1 mo.	Does not see it.
3 mo.	Transient regard.
4 mo.	More prolonged fixation of regard, beginning of attempt of hand to move toward, a flare.
5 mo.	Fixation of glance; sweeping approach or out and back again abduction before adduction.
6 mo.	Slightly different approach from side, grabbing motion.
7 mo.	Thumb in when first moved toward object. Thumb gradually becomes exerted, but not used at once. First grasp is palmar with tight squeezing. Does not know to let go.
9 mo.	Index finger takes precedence though does not grasp.
10 mo.	Begins to grasp sides between thumb and index finger.
12 mo.	Regular pincer prehension.

Hand Position

to 8 wk.	Thumb mostly in hand.
12 wk.	Thumb mostly out of hand.
7 mo.	Thumb inverted, but not used.
10 mo.	Thumb lateral grasp.
12 mo.	Thumb pincer grasp.

The Relation of Environmental Pressures to the Development of the Child's Personality and Habit Patterning

Jean Walker Macfarlane

The first paper presented in this symposium has placed its emphasis upon the implications of the physical make-up and growth to personality development in children. I have been asked to present material which will emphasize the effects of environmental pressures upon personality and habit patterning. I shall restrict myself very largely to that psychologically potent area of the child's environment —his home and, more particularly, his parents and his siblings. I shall try to indicate where the pedintrician, who has more contact with the family during the formative preschool years than any other trained person, has peculiar opportunities for effective influence.

Obviously, in understanding any given child, one cannot separate his behavior from his constitution, health history, degree of development, or from the environment to which he has had to adapt himself in the past or with which he is now confronted. Any attempt to institute a program of training or therapy which fails to take n careful inventory of the particular psychobiologic organism with its own peculiarities of equipment or which fails to take a detailed inventory of the per-

sons and situations to which this child has to adjust, is doomed to ineffectuality. Behavior, whether it be a source of parental pride or whether it sends its owner to a child guidance clinic, is the resultant of what the youngster has had and now has to adjust *with* and what he has had and now has to adjust *to*. This is such an obvious and basic and simple truth that, caught as we are in our own technical complexities and refinements, we are apt to lose sight of the basic mechanism of biologic life—namely, the immediate attempts of the organism in disequilibrium to get into activity which restores equilibrium. The *need* for adjustment depends upon disequilibrium; the *manner* of adjustment or behavior depends upon the structure of the organism and the nature of the stimulation from which it seeks relief.

If we could always remember to think of the youngster's behavior in terms of both his equipment and the pressures put upon it, we should avoid, I believe, many fancy and footless excursions. If we could remember that his behavior represents *his* adjustive devices to seek equilibrium, to satisfy his needs, to placate his pressures, I believe we would be on the road to discovery of what needs and pressures were driving him and be able to help him to more socially acceptable and more health conducive patterns.

Many of the ideas and most of the material I shall present have grown out of a ten-year guidance study at the Institute of Child Welfare at the University of California.* The group consists of a normal cross section of 252 children and their respective families. These families were arbitrarily selected upon the basis of every third birth in Berkeley, Calif., from January, 1928, to July 1, 1929, and have been followed for ten years. They were divided into a guided and an unguided group. This study differs from other clinical studies in two respects. First, it gives us material on a normal group, and its findings can, therefore, be taken as a check upon findings from therapeutic clinics. Second, its method has been cumulative observation of developing patterns, both adjustive and maladjustive, rather than the usual one of trying to unravel the antecedents of already developed "problem behavior." It gives us, we feel, a more accurate body of information than that obtained retrospectively.

The major objective of this research has been to portray the course of development of numerous specific aspects of behavior—both adaptive and maladaptive patterns—and to investigate relationships between behavior patterns and such factors as health, physique, physical growth patterns, the child's intelligence and rate of intellectual growth, socio-economic measures, and other aspects of his physical environment, personality characteristics of parents and siblings, interpersonal relations within the home, and school environment and progress. Systematic cumulative data have been obtained in all these fields.

A second objective has been to study the frequency, persistence, severity, and dynamics of "problem behavior" and "personality problems" in a cross-section group and to discover the relationship of earlier to later behavior. Without a careful comparison of later and earlier behavior, we are apt to overemphasize some behavior as serious which the child may easily outgrow and to overlook behavior not troublesome at the moment which may lead to serious neuroticism or maladjustment later.

A third objective is to check therapeutic effectiveness by comparing a group subjected to guidance procedures and a group equated but unguided. Too often it has been assumed without a critical check that the intervention of guidance procedures has brought both good and permanent results. And far too often, I believe, such people dealing directly with children as the teacher or pediatrician have been made suspicious or antagonistic by the ex-cathedra dogmatism of the many opposed schools of psychotherapy.

*Macfarlane, J. W.: Studies in Child Guidance: Methodology of Data Collection and Organization. Child Development Monograph (in press).

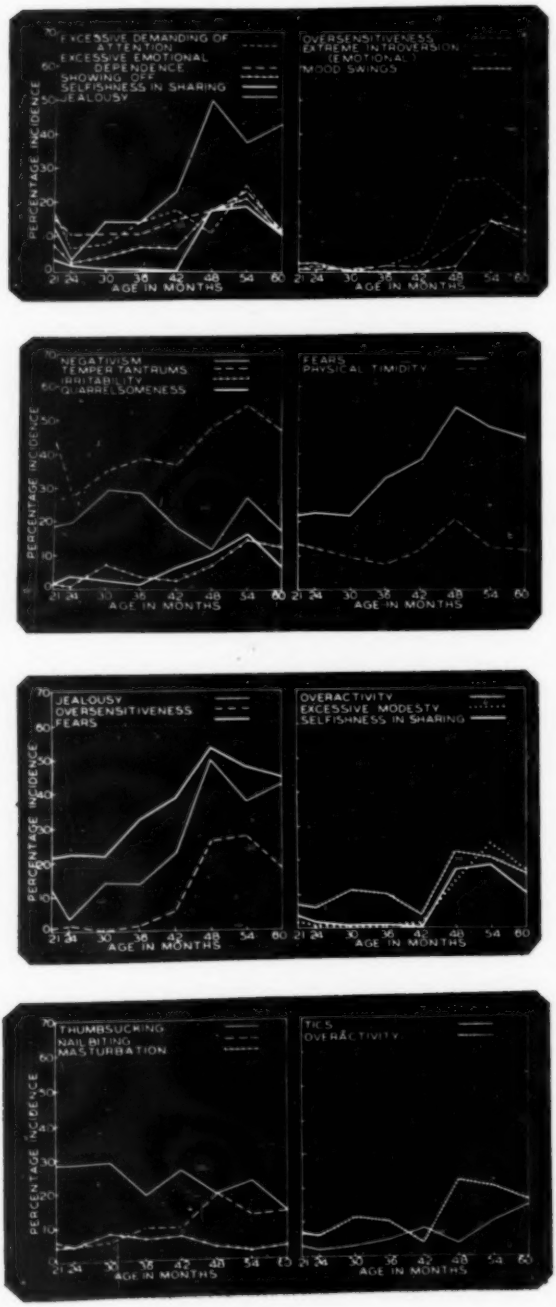

Chart 1.—Age changes in incidence of problem behavior.

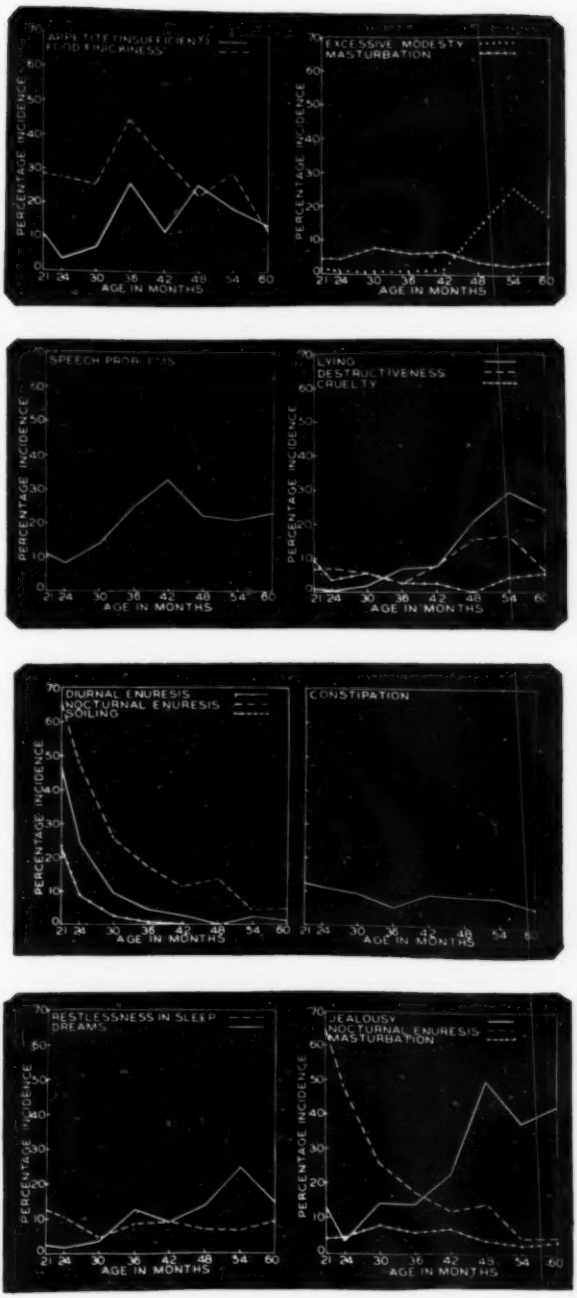

Chart 1—Cont'd

After this lengthy preamble, I shall present findings, first, on the frequency in a normal sample of behavior which has been labeled as "problem behavior"; next a very few findings on the relationship of physiologic status to certain problems; and for the remainder of the paper, I shall discuss the relationship of personality and behavior patterning in the children to their family situation.

We have found that no normal child is completely free of adjustive devices that are labeled as "problem behavior," the average number varying during the preschool years at any one age level from four to six per child.

Chart 1 shows the frequency figures for various patterns. It can be seen that frequency was found to vary with age for most problems. Soiling, diurnal and nocturnal enuresis decreased with age and were eliminated in the order named, whereas constipation showed no trends with respect to age. Masturbation and restlessness in sleep showed no trends with age for the preschool period. Thumb-sucking decreased as nail-biting increased. Tempers, fears, jealousy, oversensitiveness increased to around 4 or 4½ years and then began to subside. Since temper tantrums, fears, and overt jealousy occur at the preschool level in more than 50 per cent of our children, they cannot sensibly be regarded as neurotic behavior as so commonly assumed, but rather as evidence of tension or adjustive devices. If they continue, however, and become fixed habits of response, they may become thoroughly inappropriate patterns to meet situations outside the home and become in themselves additional hazards in adjustment.

There is some statistical evidence that certain patterns tend to fall into clusters.* For example, at 5 years *quarrelsomeness, mood swings, negativism, irritability, temper tantrums, jealousy,* and *competitiveness* show similar degrees of relationship to other problems. This cluster of problems suggests a labile or disturbed organism. Another cluster suggestive of a child who is psychologically withdrawn and below par physically includes the following problems—*withdrawal, introversion, submission, shyness, somberness,* and *excessive reserve.* These problems are all closely associated with *underactivity.*

So much for the actual behavior patterns we see in the preschool child. We should bear in mind that the same symptomatic behavior may have totally different meanings and causes. Just as the child full of insecurity or anxiety may seek relief in a variety of behavior patterns, so likewise do we find that the same behavior may be symptomatic of various drives or pressures or conflicts. Unless we discover the meaning for a given child, we do very little, in spite of altering the symptomatic picture, to relieve the tension which has given rise to such behavior. Some symptoms, it is true, are worth altering because in and of themselves they are handicapping. Some behavior consists merely of old habitual patterns, once serving a useful purpose no longer present, but many so-called maladaptive patterns have real adaptive purposes in meeting needs and solving conflicts. The important therapeutic attacks must be in terms of help in solving the conflict, or if that is not possible, at least to get it on a conscious basis so that more suitable patterns of compensation may be employed.

Now for a brief statement of the relationship of behavior patterning to physical condition or constitution. Clinically, of course, we found many complicating physical conditions interfering with or shaping personality development. For example, youngsters with hives, eczema, or any irritating physical condition were more apt to develop tempers and overactive responses than youngsters free of such irritations. Shyness and somberness found more recruits among children with low energy levels. The undersized boy, the oversized girl, even at the preschool level, showed evidence of building up defensive patterns either of shy withdrawing or aggressive showing off.

*The method of cluster analysis will be described in a forthcoming publication by R. C. Tryon. This cluster analysis was done by Miss Claire Myers.

A preliminary statistical analysis of the relationship of physiologic condition to specific problems shows interesting results. For the preschool period certain problems appear much more largely related to poor physiologic status than do others. For example, elimination problems, eating difficulties, nail-biting, over-dependence, and negativism in both the guided and unguided groups, and for the different age levels, occur more frequently in children with poor rather than good physiologic status. Those problems, therefore, occurring frequently in children below par should be peculiarly the problems of the pediatrician, and techniques of effective intervention in modifying them, important to his equipment. Problems which occur more frequently in children of better than average physiologic status are thumb-sucking, overactivity, lying, and fears. Tempers and jealousy occur as frequently among children with good as among children with poor physiologic status. These problems seem much more related to psychologic factors than to physiologic ones.

Now as to factors within the home and their relation to the child's adjustive patternings: It seems to be the irony of fate that children pick out the areas of the parents' greatest concern in which to develop disturbing habits. This does not appear to be a matter of chance or bad luck, but rather an inevitable result of circumstances. This adjustable organism, the child, confronted with the relentless pressure of training, quickly sounds out the areas of his parents' vulnerability and makes the most of them. If you are a child of 4 years confronted with adjusting to a younger sib, who is using your crib, your toys, your clothes (outgrown, but still your own), and further if this younger sib is running off with your mother's attention and affection, which were formerly your own exclusive possessions, you seek in desperation to get back the security and equilibrium which were once yours. The nature of your attempts to get back this security will depend upon your own eccentricities and upon those of the personalities in your situation. If you have a low tolerance for fats, a mother who has been made sensitive to health by a history of tuberculosis in her family, and a father who has been sensitive about his small stature and blames his stature not upon his genes but upon his early eating habits, your chances for seeking security through fussy eating habits are very high. You quickly see, if you have a normal I.Q., that the anxious solicitude you obtain for *not* eating brings 100 per cent attention for yourself and 100 per cent parental ignoring of your intruding sib. If you have the same problem, namely this sib who has disturbed your equilibrium but parents who are not concerned about health or your eating, fussing over eating does not yield results. But if your parents are very upset when you sob, or are afraid, or wet your bed, or have a temper, or tell a lie, you learn to pick the tool most effective with them whether it be lies or painful honesty, or whether it be wistful tears or heavy demonstrativeness, or merry showing off or contrite expressions such as, "I'm sorry, papa, I did wrong."

In any area where the parent is emotionally conditioned by past experience or present situation to anxiety, he is apt to be unwise or exploitable or uncertain. It is a common clinic experience to see a mother who can take in stride and handle objectively the trial and error behavior of her child involving tempers or matters of health but who goes all to pieces over masturbation. Another may handle quite objectively the matter of sex experimentation on the part of her child and be disturbed if he is afraid of the dark or tells a lie. We all have our peculiar areas of hyperreactivity and anxiety, and these areas in parents are an extremely important factor in understanding the particular behavior developed by the child and must be appreciated by anyone attempting a program of modifying behavior which is unhygienic or handicapping. One gets no place by labeling the child spoiled or neurotic and the parents fools, as we are all apt to do when we give advice which is not followed and which may not work if it *is* followed. (I should

state as an aside that nothing can give a greater sense of humility than a long term follow-up study which places in your unobstructed view the fruits of your own optimistic advice faithfully followed.) One learns in time that most advice is either bad or futile and that the only fruitful approach in behavior and habit modification is understanding in the hands of those who do the training.

May I refer to the 4-year-old child mentioned above who was an extremely fussy eater and who was thrown off equilibrium by the arrival of a sibling and the reduction in attention from her mother. Knowledge of her low tolerance for fats should lead to effective modification of her diet, but the chances of her stopping finicky eating habits would be very small if they were bringing her undivided and solicitous attention from her parents. The parents would have to appreciate the basic need these finicky habits were being used to satisfy and to find more happy ways of satisfying this need. Further, the parents would have to be led to an appreciation of their own anxiety about health and eating. Knowledge of the sources of our overanxious attitudes helps to desensitize us. The mere fact of getting a history of previous experiences with illness and of attitudes toward health and knowledge of how attitudes have been built up out of worries gives to the person overconcerned a sense of understanding which does much to lessen anxiety. And even where overconcern remains, the person with this understanding can much more effectively and consciously compensate for it. Twenty minutes devoted to a sympathetic questioning of a mother about the health and attitudes toward health in her family as a child can do much to give her a clear picture of why she has her present attitudes. In fact it can often do more good than twenty minutes devoted to the details of how to manage the child's eating habits. If she is freed, by understanding, from anxiety, she is free to be sensible herself about working out details. If she is given detailed instructions and merely told she is overanxious, she may become even more tense, and her overconcern may completely nullify instructions followed to the letter.

Let us look in more general terms at some of the statistical findings from our ten-year study. With as many forces, internal and external, impinging on the growing child, we cannot expect to find any one environmental factor which will explain complex behavior. We do find, however, that certain aspects of the home stand out as considerably more important than others. For example, the marital adjustment of the parents, whether it be a hectic and straining one or a liberating and supporting one, has much more bearing upon the child's behavior, at the preschool years at least, than any other factor in the home, including education and standard of living. Where there has been an uneasy and discouraging relationship between the parents, certain patternings in the child flourish. Of the ten most frequent problems in our preschool group, eight showed higher and more consistent statistical relationships to marital adjustment than to any other factor studied. Food finickiness, overdependence, attention-demanding, negativism, temper tantrums, and diurnal enuresis are found more largely and more consistently in homes where a straining or inharmonious parental relationship exists. Nocturnal enuresis and thumb-sucking during the early preschool years occur more frequently in children whose parents have a harmonious relationship.

So it is obvious to anyone working in a guidance clinic for preschool children, particularly where the child is spending a major portion of his time in his home, that the home is far more often the problem than is the child. Yet frequently this fact is cited in criticism of the parents rather than in terms of a sympathetic and active program for increasing understanding and reducing strain between them. In certain cases, to be sure, the marital relationship is so snarled or offers so few potentialities to mutual support that little can be done about it. Yet in a very sizable proportion of cases, real help can be given. It is interesting to note that

in a detailed questionnaire given to our parents concerning wherein the clinic had helped or failed to help, had reduced tension or increased it, over 50 per cent of this unselected sample cited help on marital adjustment as the clinic's most significant contribution.

Since marital adjustment is so important to healthy habits in the child, what can be done about it? and how? and by whom? What can be the pediatrician's contribution to the increase of parental understanding and to the reduction of marital strain?

Let us exclude from our discussion those parents with disorganized personalities, the highly neurotic or psychotic parents who need protracted psychiatric attention and care, and look at the large run-of-the-mill group whom we have found vitally interested and capable of increased understanding. What for them are common sources of strain and bafflement, even in the face of undeniably genuine affection for their marital partner?

In our group, we found that, while on the whole similarities of economic background and educational attainment were the rule in marriage, this was not true with respect to temperament. The reserved dignified person with a marked sense of privacy tends to marry the outgoing extroverted person whose major satisfaction comes in shared experiences. Apparently a large element of romantic allure lies in a temperament different from one's own, and yet this different temperament may be baffling to understand and to adjust to. We erroneously are prone to interpret the other person's feeling in terms of how we would feel if we were behaving in a similar way. It is easy for a demonstrative outgoing wife to feel that she is more attached to her reserved husband than he to her, whereas in many cases it is the reserved person who is more exclusively and thoroughly attached and aware of it but too inept or reserved to show it. A realization of this fact does much to reduce uneasiness in the baffled extrovert.

Another adjustment which, without understanding, may bring strain grows out of differences in attitudes and practices built up from childhood. Take, for example, the management of finances. The woman who was fond of a father who paid cash as he went, carried heavy insurance, turned his pay check over to his wife to manage is bothered by a husband who runs many dragging charge accounts, who thinks heavy insurance foolish ("Live merrily as you go along," says he), who manages the salary check and doles it out as she needs it. Not only is she unaccustomed to new practices and therefore thinks they are wrong, but a disquieting feeling arises that, if he were really fond of her, he would pay cash, etc., because these were patterns practiced by the one man who was fond of her in her childhood. The husband, following the practices of his own father, may have no notion that she is bothered or, if he senses her disapproval, catalogues her as temperamental or fussy. We have found that a review of practices and attitudes of the respective families in regard to finances, education, health, disciplinary techniques, religion, recreation, etc., did much to give each marital partner a realization that the practices of each were outgrowths of early attitudes and had nothing to do with lack of affection or confidence in the other. As one mother put it, "I never realized before that to him what he did was natural. I just thought he was stubborn and inconsiderate and I guess he thought I was, too."

A mutually satisfying sex adjustment brings enough support, when it involves affection, to make the adjustments just mentioned seem relatively minor; but, if either of the pair is thwarted or feels inadequate to meet the drives of the other, all sorts of minor items are saturated with the strains projected from erotic tensions. Sometimes a poor sex adjustment grows out of poor health and low energy level, sometimes out of worries over possible pregnancies—both of which are problems within the scope of the physician to help. We frequently found that adequate contraceptives not only restored erotic equilibrium, but made other pressing

problems evaporate. Sometimes a poor sex adjustment was caused by ignorance of differences in sexual reaction times and rhythms or ignorance of techniques. Far more often it was related to psychologic hurts, conflicts, or preoccupations. A father reported that with the arrival of the children, the wife was so preoccupied with their care that he felt excluded from a closed corporation; and the wives simultaneously reported that, since the children came, their husbands seemed less interested in them. Often each was hurt and felt less important to the other with the new function of parenthood altering their functions to each other. Often we found, especially with young parents not yet established financially, that the cost of caring for children had precluded almost any recreation together which had formerly been a major source of fun and liberation.

From this sketchy presentation, I am sure you can see many instances in which you can be of help, and instances where you have not the time nor perhaps the technique of skillful interviewing necessary to a nondamaging intervention in the realm of emotionally laden husband-wife relationships. But you can give sympathetic support, urge tolerance of persons with personalities and experiences different from one's own, inquire about rest and recreation for the parents, urge each of them to take stock of his own needs and those of his partner, and help them to better regimes for themselves and to adequate contraceptive advice. The least you can do is to avoid criticizing them and adding to their tensions. I hope the time will come when all medical schools will equip their pediatric interns with techniques of taking pertinent inventories of that highly potent area of the child's environment—the adjustment of his parents.

Closely related to marital adjustment as an important factor in the child's environment is parental agreement or disagreement on discipline. No two parents agree completely on disciplinary techniques, partly because they as children were subjected to different techniques and modes of training, and partly because the same techniques are not appropriate to different personalities. A woman cannot act like a man, nor a man like a woman, and be convincing to an offspring. A quick-moving domineering person cannot pretend to be sweet and mellow merely because his spouse is and wants *him* to be, without adding to his child's confusion. Where parents have not accepted differences and where real strain and friction over discipline exist, we find that the child caught in this strain and indecision is more likely to make aggressive demands for his parents' time and attention through tempers, showing off, soiling, and enuresis than the child free from such parental strain. Here, again, knowledge on the part of parents that they cannot be expected to use identical techniques successfully, often results in each one's using methods more appropriate to his own experience and personality; this not only offers the child a less ambiguous learning problem, but reduces strain for the parents as well.

Parent-child adjustment is another important aspect to consider. Particularly crucial is the mother-child relationship, since usually the mother spends much more time in caring for children. We find many mothers hectic, uncertain, and uneasy over the details of raising a child, running frantically to P. T. A. meetings, parent education groups, poring over the latest undigested words from child psychology, newspaper columns, radio broadcasts, and mental hygiene propaganda, anxiously seeking advice and authority. She may be hectic because of conflict in herself or because of her domestic situation to which her child ties her; or she may be baffled by an emerging personality so unlike her own, or so like her brother's with whom she never got along, or like her husband's which she cannot either understand or accept. She may be bothered because she is reliving through her child the tensions of her own childhood. Regardless of the causes, and they are myriad if she is ambivalent and uneasy and strained in her relationship to her youngster, she offers an environment making for increased likelihood of certain unfortunate re-

sponse patterns in him. Here, again, a therapeutic program for the child must be largely in terms of desensitizing the mother. Sometimes, with proper techniques, this can be a simple matter; often it means a fairly long program of bolstering, of helping her to conscious appreciation of the basic causes of her tension, or of the need for more recreation, or the cultivation of new interests or revival of old ones which formerly gave her support and satisfaction. What she does *not* need is criticism, for she is full to the brim of self-depreciation.

I am sorry time precludes a discussion of intersibling relationships and their effects on patternings. I shall list one or two outstanding impressions. The older child has a complicated problem to handle by the arrival of a younger sib. We have found that a frequent result of the destruction of his monopoly of affection leads to uneasy patternings in affectional relationships. He is more likely to have difficulty in establishing easy, friendly and intimate relationships with people, even outside the home. Much can be done, of course, later to give him insight. Much can be done to forestall disruption of complete dependence by training to independence before the arrival of a second child. A younger child may be much more secure in affectional response and easier in social relationships, but he is confronted with a situational adjustment, too. His older sib is a person always, because of greater relative maturity, placing him in an inferior role with respect to achievement. If this older sib is a boy, the second child may constantly feel inadequate intellectually and physically. If this older sib is a pleasantly maternal girl, the younger child may continue most of his life hanging on to childish patterns which attract older sister substitutes and retard emotional maturity.

In conclusion, may I repeat that, although this paper has laid its emphasis on environmental pressures, some children are much more sensitive and much more adversely modified than others by environmental strains. The hypersensitive and overreactive organism may give much more exaggerated reactions than a sturdy, less irritable organism, but even a sturdy organism can become an irritable one with long-continued strain. In understanding behavior, one needs to know the organism and its capacity to absorb pressures, and one needs to know the nature of the pressure to which organism must adjust. In the early years of laying down emotional and behavioral patterns which become the warp and woof of later personality structures, the most important environmental area is the family with its various interacting personalities. Since this is true, any program of guidance must take stock of these personalities and their interrelationships with the same careful scrutiny that goes into an adequate physical examination and diagnosis. This takes time and background and special techniques. Some problems are so complex that adequately trained and experienced specialists are the only ones who can successfully handle them. There remains a very sizable group of problems peculiarly the responsibility of the pediatrician, for, if he does not handle them, they will be left untouched in a vast majority of cases as he is the only trained person who has an opportunity to discover them. I look forward to the time when a pediatric history will include what the child has had and now has to adjust *with* and what he has had and now has to adjust *to*.

Rating Scale for Family Situation
(*Guidance Group*)

Nervous Stability—Mother, Father

1. Exceptionally stable, a very even keel in face of most trying circumstances
2. Markedly above average
3. Above average
4. Average, occasional upsets in response to trying situation
5. Excitable, tense, overactive, sullen, extroverted, exaggerated reactions

6. Neurotic, alcoholic, prostitutes, very eccentric personalities which interfere with work or social adjustment
7. Psychotic episodes, or equivalents

Marital Adjustment

1. Exceptionally happy adjustment, frankness, affection, interests, agreement over finances, discipline, sex, etc.
2. Above average, many satisfactions, no disruptions
3. Normal adjustment, occasional upsets, real satisfaction
4. Working arrangement difficult, more or less chronic tension
5. Extreme conflict; separation, divorce

DISCUSSION

DR. OSCAR REISS (LOS ANGELES).—We may define the term "mental hygiene" as a technique which enables the growing individual to stand firm under the growing pressures of life. We are yet in the stage when a child is brought to a pediatrician for physical complaints. A careful history will bring out psychologic disturbances. We must evaluate and separate the trivial from the serious, we must acquire a vast, wide, and discerning knowledge of the things which occur to children and how they influence the child.

DR. C. D. SWEET (OAKLAND, CALIF.).—Fear is important. We must teach mothers that all humans have fear. "A brave man is a coward who does not run away." It takes eight years to learn a little arithmetic, yet we expect a child to learn courage in a week.

CHAIRMAN LAYMAN.—This opens the wide vista of what is wholesome fear.

DR. SWEET (OAKLAND, CALIF.).—A neurasthenic is a person who exaggerates what he has more than his neighbor.

DR. S. H. ASHMUN (DAYTON, OHIO).—It is essential to make sure there is no physical basis for these upsets in children.

CHAIRMAN LAYMAN.—It is very difficult to separate functional from organic.

DR. HORTON CASPARIS (NASHVILLE, TENN.).—Every child is a problem. If we are too enthusiastic, we will make a greater problem out of a normal child. We must agree that the term "mental health" is preferable to "mental hygiene." Hygiene suggests dirt rather than behavior. Mental health must be considered as resting on the same health basis as physical health. Granting this, it follows that the pediatrician must assume the responsibility for the behavior problems of childhood as well as for the physical. The child guidance clinic gets the child only after he is already in trouble. The important problem is to reach the child before his troubles begin, and this the pediatrician must take upon himself. Only in this way will it be possible to turn the tide of mental ill health which is certainly on the increase.

No one knows what normal behavior is. The first necessity, however, is the ability to face life adequately. The child, as he lives and meets new problems, asks for and strives to obtain comfort and pleasure. In this, as in other respects, child psychology is the same as general psychology. The problems of the child are fundamentally the same as those which confront the adult. Mentally adequate individuals have grown up since the beginning of time by the observance of certain basic laws. First, the child must have the courage to face life without being hurt by it. He is born with a certain amount of this essential quality, but it is equally true that he may dissipate it and become bankrupt. In fostering the development of courage in the young, it is important to remember that, as with mus-

cular development, it improves with use. A baby cries to express his disapproval of a situation confronting him and for help in evading it. He should be taught to face his difficulties by giving him only the attention he requires, not by sympathizing with him. Teach the child that he does not get things by crying, but by being good. An oversympathetic attitude towards the child tends to decrease his courage and increase his demand for attention.

Law-observance, respect for the rights of others, is the basis of all laws, written and unwritten. It is also the foundation of a good personality. It may be trained into a child, but it is a slow process. It is best learned by imitation.

The third essential for the mentally adequate child is a sense of security. He must be taught, however, that any security which is not acquired by his own efforts is demoralizing. In other words, he must stand on his own feet.

These are the three main foundation stones of a satisfactory existence.

The normal child is born with an urge to do things. To continue in this, he must have mental nutrition, a feeling of accomplishment. His efforts, however, should be kept within the limits of his capacity. He will not continue to put forth this effort without some sense of accomplishment. Each new act accomplished stimulates him to do more. Recognition of his accomplishment is to him as is his pay check to the laborer.

Most of the problems of childhood are tied up to these factors or basic principles.

How do you make a child mind? The answer is to make it unpleasant not to mind, and the unpleasantness must follow disobedience immediately. Telling the truth is more or less natural and should be made pleasant.

QUESTION.—Do you have much trouble with stepparents?

DR. MACFARLANE.—We are very likely to have trouble in families with stepchildren if the child is not told of his status in the family and finds it out for himself. He should always be made aware of the true situation by a sympathetic informant. Trouble is not likely to arise when he is told early. Mothers should be less sensitive to opposition on the part of stepchildren.

CHAIRMAN LAYMAN.—Every child wants to feel that he belongs. It gives him the necessary sense of security. If he finds out early that he belongs in the family, later complications will be avoided.

DR. MACFARLANE.—The adolescent child is naturally critical of his parents. If the family situation is explained to him early in childhood, this critical spirit will be less acute than will be the case if he comes to this knowledge later.

QUESTION.—Is it advisable to adopt a child into a family as a companion for another child?

DR. MACFARLANE.—This must depend entirely on the attitude of the parents to both children. It would be undesirable if the parents did not accept the second child on the same basis as the first and endeavor to give him the same affectionate care.

DR. LANGLEY PORTER (San Francisco).—There is no such thing as a child entity. He is the result of the reactions between many things. There are no such things as good and bad parents and children. The biologic unit is the family. The family is an institution for the development of a family. You do not believe in the value of admonition or reproof? You believe in establishing a form of behavior pattern which the child can imitate?

DR. MACFARLANE.—Yes.

QUESTION.—How is the pediatrician to acquire this information as to family relations so necessary to the solution of these problems?

DR. MACFARLANE.—It is almost impossible in private work. We may be able in time to simplify the technique.

CHAIRMAN LAYMAN.—I think the main thing is to recognize that there is some ultimate driving motive back of every word and attitude, and very definite patterns of behavior result, much more typical and less complicated than in adulthood. If the approach is proper, parent cooperation is usually forthcoming. The important thing is to know that *there are* such problems.

QUESTION.—Are the endocrines a factor in child behavior?

CHAIRMAN LAYMAN.—Yes, there is a great field which will be opened in time in the field of endocrinology. This is perhaps the next step.

DR. PORTER.—The pediatrician who goes in and out of the homes of these children is in a superior position. He is the only person able to solve the problem ultimately. But it has to be done in the home not in the office.

EUGENE H. SMITH, Ogden, Utah
MARGARET M. NICHOLSON, Washington, D. C.
Secretaries

News and Notes

The following have been certified by the **American Board of Pediatrics**, following the examinations in New York City in April, 1939.

Dr. Eleanor L. Adler, Yonkers, N. Y.
Dr. Charles V. Baker, Brooklyn, N. Y.
Dr. Harry F. Becker, Battle Creek, Mich.
Dr. Sidney Blumenthal, New York, N. Y.
Dr. Sydney M. Borowsky, Philadelphia, Pa.
Dr. J. Edmund Bradley, Baltimore, Md.
Dr. Elizabeth Brakeley, Montclair, N. J.
Dr. Henry Cohen, Brooklyn, N. Y.
Dr. Irvin J. Cohen, Brooklyn, N. Y.
Dr. Harold William Kinghorn Dargeon, New York, N. Y.
Dr. Curt Herman Falkenheim, Rochester, N. Y.
Dr. George J. Ginandes, New York, N. Y.
Dr. Jerome Glaser, Rochester, N. Y.
Dr. Harry H. Gordon, New York, N. Y.
Dr. John E. Gundy, Port Chester, N. Y.
Dr. Albert Scott Harden, Jr., Maplewood, N. J.
Dr. Edwin Adams Harper, Lynchburg, Virginia
Dr. Fred Herbert Harvie, Belleville, Ontario
Dr. David Hauptman, Brooklyn, N. Y.
Dr. Robert C. Hood, Washington, D. C.
Dr. Frank Jay Jocobson, Providence, R. I.
Dr. Edmund N. Joyner, III, New York, N. Y.
Dr. Herbert Richard Kobes, Augusta, Me.
Dr. Irving Kowaloff, Brooklyn, N. Y.
Dr. Jacob D. Leebron, Philadelphia, Pa.
Dr. Charles Joseph Leslie, New York, N. Y.
Dr. Harold Levy, Brooklyn, N. Y.
Dr. Henry H. Lichtenberg, New York, N. Y.
Dr. John Alden Lichty, Jr., Rochester, N. Y.

Dr. Harold Lipschitz, Brooklyn, N. Y.
Dr. Joseph P. Michelson, Brooklyn, N. Y.
Dr. William Paris, Chelsea, Mass.
Dr. Milton Rapoport, Philadelphia, Pa.
Dr. Solomon Rauch, Brooklyn, N. Y.
Dr. Rudolf L. Roddy, Philadelphia, Pa.
Dr. Irwin Schiff, Brooklyn, N. Y.
Dr. Richard Schorr, New York, N. Y.
Dr. Joseph Schwartzman, Brooklyn, N. Y.
Dr. D. William Scotti, New York, N. Y.
Dr. Lydia Shapiro-Mindlin, New York, N. Y.
Dr. Eli Y. Shorr, New York, N. Y.
Dr. Louis Spekter, Hartford, Conn.
Dr. Martin L. Stein, Brooklyn, N. Y.
Dr. Harry Strongin, Brooklyn, N. Y.
Dr. Clifton C. Taylor, Bridgeport, Conn.
Dr. Abram S. Tepper, Far Rockaway, N. Y.
Dr. William J. Turtle, Boston, Mass.
Dr. Anna E. Wagner, Brooklyn, N. Y.
Dr. Virginia Travell Weeks, Brooklyn, N. Y.
Dr. Daniel A. Wilcox, Mount Kisco, N. Y.

The following were certified following the examination in St. Louis in May.

Dr. Alexander John Alexander, Lexington, Ky.
Dr. Ralph Newton Barlow, Logan, Utah
Dr. Jack Basman, Charleston, W. Va.
Dr. Jack Chesney, Knoxville, Tenn.
Dr. Peter G. Danis, St. Louis, Mo.
Dr. Mark W. Dick, Grand Rapids, Mich.
Dr. Clayton Thomas Joseph Dodge, East Cleveland, Ohio
Dr. Ralph E. Dyson, Minot, N. D.
Dr. A. R. Eveloff, Springfield, Ill.
Dr. Theodore Herzl Goldman, Los Angeles, Calif.
Dr. C. Jack Harrison, Chicago, Ill.
Dr. Stanley L. Harrison, St. Louis, Mo.
Dr. H. Sidney Heersma, Kalamazoo, Mich.
Dr. Frederick A. Jacobs, St. Louis, Mo.
Dr. Joseph Cabell Jaudon, St. Louis, Mo.
Dr. Reuben I. Klein, Chicago, Ill.
Dr. Ralph Hess Kunstadter, Chicago, Ill.
Dr. Matthew M. Lewison, Chicago, Ill.
Dr. J. Keller Mack, Louisville, Ky.
Dr. Thomas Myers, St. Paul, Minn.
Dr. George T. O'Byrne, Tyler, Texas
Dr. Maurice C. O'Hern, Chicago, Ill.
Dr. Henry George Poncher, Chicago, Ill.
Dr. Alwin C. Rambar, Chicago, Ill.
Dr. William H. Reals, Chicago, Ill.
Dr. Roland Boyd Scott, Chicago, Ill.
Dr. Mandel Lawrence Spivek, Chicago, Ill.

The **Southern Pediatric Seminar** will hold its 19th Session at Saluda, N. C., July 24 through August 5, 1939.

Information about the courses may be obtained from the registrar, Dr. D. Lesesne Smith, Saluda.

The **Academy Committee on Mental Hygiene** has arranged for a series of special articles on important topics in the field of mental hygiene. The first of these, "Family Relationships," by Dr. F. H. Allen, Director of the Philadelphia Child Guidance Clinic appears in this issue. Other authoritative articles in the series will be published in the JOURNAL during the coming year.

Books Reviews

The Throat Distemper of 1735-1740. Ernest Caulfield, M.D., New Haven, 1939. Published for The Beaumont Medical Club by Yale Journal of Biology and Medicine. Limited Edition, pp. 113.

An epidemic of "throat distemper" swept through New England and the adjacent Middle Atlantic States from 1735 to 1740. Starting with the outbreak in Kingston, N. H., the author traces the spread of the "epidemic." As the result of painstaking research and study of contemporary descriptions, mortality records and church archives, Dr. Caulfield is led to the belief that in reality two diseases played a part in the epidemic. One with a frightfully high mortality rate he thinks was diphtheria, while the other with a lower case fatality rate was scarlet fever.

The volume gives a vivid picture of the medicine of that period. It is illustrated by cuts of medical pamphlets, and religious broadsides urging the population to turn from sin so that the disease might be averted. The descriptions of the disease by Douglas of Boston, and Dickinson of New Jersey are of particular interest. The monograph is not only an important contribution to the history of medicine in America, but is delightfully and entertainingly written, and would be thoroughly enjoyed even by those who have no particular interest in medical history.

Studies from The Center for Research in Child Health and Development, School of Public Health, Harvard University. Monographs of the Society for Research in Child Development. Vol. IV, No. I, Serial No. 20. National Research Council, Washington, D. C. 1939, pp. 261.

A description of the objectives, development, and organization of the Center, is followed by a report of the studies in the development of the normal child, which were started seven years ago. Part III contains a brief report of the normal pregnancy and premature birth series. Part IV, the last half of the monograph, contains a detailed study of two sisters and their families. The monograph is extensively illustrated with charts and photographs.